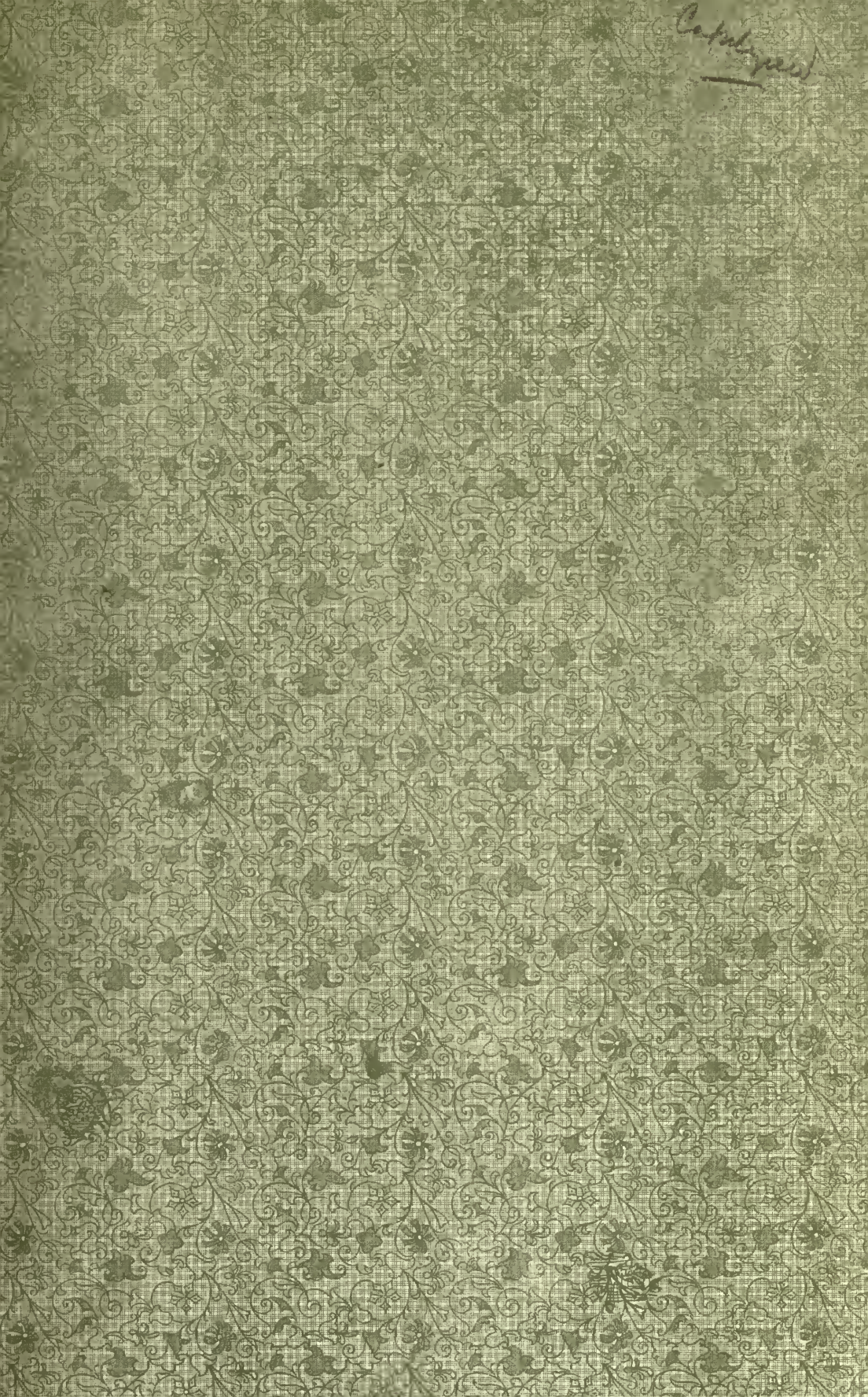


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Med.
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TRANSACTIONS

OF THE

TWENTY-EIGHTH ANNUAL MEETING

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

HELD AT
WASHINGTON, D. C.
OCTOBER 16-20, 1923

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The Twenty-ninth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Montreal, Canada, September 15-19, 1924. The attention of the members is called to the ruling of the Council, that when a paper published in the Transactions is illustrated, the author must pay the total cost of all colored plates, and one-half of the cost of all other cuts. The Editor will furnish an estimate of the cost of such cuts and plates upon request.

Only those members will receive the Transactions who have paid their dues by March 1st of the year for which the Transactions are published.

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TRANSACTIONS
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JOINT SESSIONS

ADDRESS OF WELCOME.

HUBERT WORK, M.D., SECRETARY OF THE DEPARTMENT
OF THE INTERIOR.

WASHINGTON, D. C.

Mr. President and Fellow Physicians (if I may call you that after my temporary aberration): It is a very great pleasure and privilege to me, as a temporary citizen of Washington, to extend to you the hospitalities of this city. Of course I was not born and brought up in Washington—but feel I am entirely within my prerogatives when I undertake at this time to welcome this distinguished body of medical men. The semidetached position I have occupied for several years has given me a viewpoint of the medical profession and medical men which is a little different perhaps from those whose time is taken up continuously with the work and the technic of the practice of medicine.

The medical profession of this country, about 140,000 all told, has become a class of citizens by itself. The medical training that a physician gets through his daily practice is a little different from the training that comes to any other man. Really, the processes of government are founded upon the fundamental principles that you men follow in the practice of medicine. Government, of course, is thought to be of the people, by the people and for the people; but Government cannot be conducted from the outside in; it must be conducted from the inside out, and a Government official who can arrive at the diagnosis, if you please, of conditions, and trace out from that condition the symptoms which others call results in the economic world, is apparently the man who arrives at his conclusions quicker and accurately. It has been customary for a good many years past for anyone interested in Government affairs to study conditions on the outside in his own district, or over the country at large, and try to devise methods and expedients of government that would apply to these conditions. No one can realize as you men do that this is the wrong way to proceed. You would find what the trouble is and trace the results from that and remove the cause. Conditions have a great tendency to right themselves even in Government. This country, which has been disturbed—and we were all in it a few years ago—is slowly settling down and becoming more conservative and more sane, and we find the fundamental principles of government are very similar to those which the medical men apply in their everyday work. I am not a politician, and I

do not want to say anything that sounds like a political speech, but this thought may interest you. The time will come, I am sure, when the medical men will be drafted into public service more and more. It is true in the older countries. It is common practice for those countries to send medical men to represent them in this country as ministers, and I attribute it to the fact that they have a habit of mind which comes from their study and practice of medicine, which fits them for governmental positions.

You may be interested in the medical phase of my Indian Service. There are approximately 340,000 Indians in the United States, an increase of 13,500 during the last ten years. Over 100,000 of these have been removed entirely from the jurisdiction of the Interior Department, so far as their property rights are concerned.

The medical files of the Indian Office for the fiscal year, ending June 30, 1923, indicate that there are approximately 19,073 Indians suffering from active and latent tuberculosis, and 27,745 suffering from trachoma, including conjunctivitis follicularis, which is approximately 50 per cent of the total number of Indians suffering from trachoma.

The appropriation for health work in the Indian service for 1911 amounted to \$40,000, but our current appropriation carries \$370,000; we are asking in our estimates for next year \$100,000 more.

In 1888 the Indian Service had four hospitals; this has been increased to 78 at this time. Our total hospital capacity amounts to 2,222 beds. During the fiscal year there have been cared for in different hospitals approximately 16,000 patients, giving a total of about 486,000 hospital days.

In 1895 we had 74 physicians in the Indian Service; we now have 208. In 1895 we had eight nurses; we now have 105. In 1895 we had seven other hospital employees; we now have 100. In 1895 we had three field matrons; we now have 87. We also have seven travelling dentists, who travel from one jurisdiction to another in their respective districts and perform dental work for the pupils in the schools and also for the reservation Indians.

The Public Health Service has cooperated closely with the Indian Service, and in 1913 submitted a report to Congress on "Contagious and Infectious Diseases among the Indians."

The Red Cross a year ago detailed three of its nurses to our Service, who have been working among the Indians since then and have rendered valuable service.

The National Tuberculosis Association made a careful investigation of the health conditions among the Indians recently, and their report is published in Senate Committee Print, 67th Congress, 4th Session.

Of course, the Indian Medical Service has been under the supervision of nonmedical officials since the beginning, and one of our working embarrassments is due to the subjugating of the authority of medical men to lay superintendents. It is a scattered, difficult, poorly paid service. Possibly our proposed reclassification of salaries may assist us some.

This meeting is in a way something of a humiliation to me. I am told that it is the largest special medical society in the world. It has been my practice on occasion for several years to make the proud boast that I was at one time the president of the largest special society in the world; but I was told yesterday by your distinguished chairman that your membership is almost 1500. 1000 was the membership of the society I speak of, the American Psychiatric Association, with a membership selected from the United States and Canada.

Specialism is expanding itself. Only a few weeks ago I was talking to a specialist in this city, a member of your society. He was reciting a very interesting case which had come to him for nasal trouble, and which he found on examination to have a beginning lobar pneumonia. I said to him, "What do you know about lobar pneumonia?" He replied, "Not much; but when a patient comes to me for special trouble I always give him a careful physical examination." There is the evolution of specialism—that comes from within out. I recall when we first heard of specialists, that it was the boast of many men that they knew nothing about any conditions except those which pertained to their own specialty. A physician who knows nothing about any part of medicine except that which pertains particularly to his own specialty does not know enough to practice his specialty.

Now, gentlemen, I know from a few minutes' observation of your program what your Association stands for. As I look down the list of former Presidents, I see the names of the most distinguished men of this country. Each name I recognize as having its place in American medicine, many of them I knew personally, and your own presiding officer, if you will allow me to say it, whom I have known intimately for twenty-five years, is one of your own best recommendations.

I welcome you to this city, to its privileges, and I would like to add a welcome from the Department of the Interior. That

just now happens to be my assignment. We occupy a building here in the city which has an auditorium in it which we offer to the Association, and if you care to come there this evening we will show you some pictures—an innocent amusement and perhaps enlightening in a way.

I have attempted to welcome you on behalf of the City of Washington, and for the Department of the Interior, and now I would like to express my own appreciation and my own particular welcome to this distinguished organization. (Applause).

ADDRESS OF WELCOME.

W. H. WILMER, M.D.

WASHINGTON, D. C.

Mr. President, Members of the Academy and very distinguished guests: I have been requested to say a few words of welcome to you on behalf of my colleagues in this city. Before doing so, may I, Sir, have the privilege of felicitating the Academy upon its wonderful record. The seed of this society sown a little over a quarter of a century ago fell upon good ground and has "brought forth fruit an hundredfold." From your first President, Doctor Alt, to the present distinguished incumbent in office, your record has been one of great achievement, and under the wise administration of your officers you have reached a tremendous numerical and scientific potentiality. Your roster, as Doctor Work has said, is the largest of all special societies in the world. This program which I hold in my hand presents the menu of a "sweet banquet of the mind." Honored and distinguished confreres from across the seas come with their contributions.

Washington is pleased that you have selected this city as your place of meeting, and hopes that you will feel that your choice has been a happy one. The National Capitol bids you a smiling welcome, and on behalf of my colleagues in this city, may I express the wish, Sir, that this meeting may be as markedly successful as those in the past, and that it may be an earnest of future meetings fraught with great scientific value and service. (Applause).

PRESIDENT'S ADDRESS.

T. E. CARMODY, M.D.

DENVER, COLO.

Since you have honored me by electing me to the highest office in your power, the subject of an address has been almost constantly on my mind. No higher honor can be conferred than to be chosen the presiding officer of the largest society of ophthalmologists and oto-laryngologists in the world.

We have many societies that have their place and are doing good work, but I am going to propose another, not American, but international, and this, our national capital, is the city from which it should spring—"The Universal Society for the Prevention of Presidential Addresses." However, since reading the addresses of my distinguished predecessors, and of the presidents of other oto-laryngologic and ophthalmologic societies, I have felt that this society which I propose should have only one member, and there should be only one address prevented. I am sure all of you will agree with me before the end of my talk.

Education has been the theme of many of my predecessors, and it seems that this society has been especially fortunate in having in its membership nearly all of the distinguished teachers of laryngology, otology, and ophthalmology in this country, and many from abroad.

This being the case, the Committee on Instruction has been able to find teachers, already Fellows of the Society, who are especially qualified to present any subject with the exception of those of a general scientific nature.

The American Academy of Ophthalmology and Oto-Laryngology is to be especially congratulated on these courses, and on the fortunate selection of the very competent committeemen who have been responsible for their success. These courses have helped to carry the center of ophthalmologic and oto-laryngologic teaching away from continental Europe and the British Isles, with all due respect to our guest, bringing it westward to our continent; and not to the eastern section alone, for valuable contributions to our knowledge and literature are being received from the western United States, as well as Canada.

Education is universal, but we have looked long to the green fields across the ocean. When I came into this Society twenty

years ago, at the first Denver meeting, I was awed by the references to European study, and while not decrying the good we have received from abroad, many of us who have not been fortunate enough to be able to refer to those clinics have possibly been more fortunate in our associations at home, professional and otherwise. During our meeting in Boston, one of our members attended a medical clinic at Harvard, and was impressed by the wonderful knowledge of the clinician. He felt that he would like to know more about where this man was educated, and so remained to talk with him. During the conversation, he inquired where he had studied abroad, and was informed that all of his training had been obtained within the four walls of that school.

The committee on postgraduate teaching of the American Medical Association has issued a comprehensive report, but there is still much to be accomplished in organizing courses to accommodate the increasing numbers who wish either complete foundation work or supplemental courses. The necessity for this work becomes apparent to the Committee on Examinations, as many present themselves who are wholly, or at least in large part, unprepared.

However, it is impractical in one year of postgraduate work to cover satisfactorily the field of oto-laryngology as it is constituted to-day, and it becomes necessary for the student to choose certain portions to which he will devote most of his energy.

It is manifestly impossible for the physician who limits himself to oto-laryngology, to say nothing of the one who includes ophthalmology, to do his own bronchoscopies and esophagoscopies, laryngectomies, thyroidectomies, and oral and plastic surgery operations, even though he may be prepared to do the work in the cervical region when complicated mastoid lesions require it—this with apologies to the general surgeon.

Examinations in oto-laryngology with certification must be established as in ophthalmology; and, as this Society is the pioneer in requiring examinations, we must lead the way.

Having launched on the sea of postgraduate teaching, we are in a position to enlarge our field so as to reach more of our members; and more especially those, who on account of great distances are unable to attend the annual meeting.

Owing to the great success of the section meetings of our sister society, The American Laryngologic, Rhinologic and Otolgic Society, and coupled with this, the marked success from a scientific standpoint of the postgraduate course sponsored by The Colorado Ophthalmological and Oto-Laryngological Con-

gress, it has come to the minds of some of us that section meetings could be conducted along these lines at various seasons in different sections of the country, i.e., a southern section could be given during the Winter months, a north Pacific or mountain section during the Summer, while an eastern section could be held in the Fall or Spring, as seems best.

These sections should be under the control of the committee on instruction, which would work with and help a local committee. The expense should be borne by this Society, with the aid of a small fee to be charged to the student. This would eliminate the possibility of the financial failure of any course, which could be equalized by the profit of another.

This, furthermore, would in no way militate against postgraduate teaching as given in the recognized centers, as it would be mainly given to the busy practitioner, who could spare only a few weeks and possibly very little money. It would, furthermore, help to disseminate knowledge of a practical nature that could not be given in short papers at a meeting of this kind.

We have, according to our directories, nearly ten thousand physicians purporting to be specialists in ophthalmology, otolaryngology and rhinology in the United States alone, many of whom never attend a medical meeting or a clinic. Some few of these may be attracted to courses as outlined, and many may receive instruction second-hand.

That we have need of constant instruction has only to be mentioned. The number of new graduates and older practitioners taking complete postgraduate instruction during any one year is only about one per cent of the total doing special work in our respective lines. For this small number the following lines from the Rubaiyat are significant :

Wake! For the Sun who scatter'd into flight
The stars before him from the Field of Night,
Drives Night along with them from Heav'n, and strikes
The Sultan's Turret with a Shaft of Light.

Before the phantom of False morning died,
Methought a Voice within the Tavern cried,
"When all the Temple is prepared within,
Why nods the drowsy Worshipper outside?"

And, as the Cock crew, those who stood before
The Tavern shouted—"Open then the Door!
You know how little while we have to stay,
And, once departed, may return no more."

Much has been accomplished either by or with the help of this Society during the twenty years of my membership. Great strides have been made in ophthalmology and oto-laryngology, both scientifically and practically through improvements in a technical way.

I wish at this time to thank our very efficient secretary, Dr. Peter, and also the various committees. Furthermore, I wish to suggest as a help to the secretary and other officers, a permanent program committee for the scientific sessions.

In closing I wish to again thank you for the honor conferred.

VICE-PRESIDENT'S ADDRESS.

HUNTER H. MCGUIRE, M.D.

WINCHESTER, VA.

I would be indeed ungrateful if I failed to express, at the opening of this scientific session, my profound appreciation of the honor you saw fit to confer upon me at the last meeting of the Academy.

Any official connection with an organization which stands first and foremost for American ophthalmology and oto-laryngology, and which already has accomplished so much toward the advancement of these sciences, not only in America but beyond our shores, carries with it a deep sense of responsibility.

In the preparation of the program of the Section on Ophthalmology, I have tried to cooperate with and assist our distinguished Secretary, and whatever merit that program may possess is largely due to his untiring efforts. My association with him during the past year has more than ever convinced me that his labor in behalf of the Academy has been an important factor in bringing this organization to its present high state of efficiency.

In the development of the specialties which we represent, America is destined to lead the world, and this organization, working in harmony with the two other great national societies, will inevitably bring about this result.

I can only thank you for giving me the opportunity, through official connection with this Academy, to take some small part in the movement for making America the leader of the world in developing the science of ophthalmology.

THE TOXICITY OF LOCAL ANESTHETICS WITH THE REPORT OF TWO DEATHS FROM BUTYN.

ALBERT E. BULSON, JR., M.D.

FORT WAYNE, INDIANA.

The recognition of the possible fatal results from the toxic effects of any of the local anesthetics is worthy of more serious attention than given it by the average physician, and particularly the ophthalmologists and the oto-laryngologists, who use local anesthetics extensively in their work. Undoubtedly there have been many deaths directly due to the toxic effects of each one of the well known local anesthetics, more particularly cocain, which never have been reported. Every operator has a justifiable hesitancy about reporting fatalities due to anesthesia, but as the result of considerable effort on the part of the committee of the A. M. A. appointed to investigate this subject, there has been collected trustworthy evidence covering forty-two deaths following the use of local anesthetics, occurring within the last three years. This does not include cases reported prior to that time. In addition to this, the committee has discovered enough hearsay evidence to justify them in believing that many more should be added to the number.

Idiosyncrasies to any of the local anesthetics is a recognized condition, and probably every operator has had experiences to prove that in the case of cocain, an idiosyncrasy is fairly common. Fortunately, the toxic effects from cocain usually give some warning, and almost invariably can be counteracted by prompt treatment. That an idiosyncrasy for butyn may be less common than that for cocain, is possible, even probable, but the case reports which I am about to submit indicate that the toxic effects from butyn come on without warning, and are not counteracted by any of the ordinary measures used in the treatment of toxic effects from other local anesthetics.

As chairman of a special committee designated by the Council on Pharmacy and Chemistry of the A. M. A. to report on the clinical use of butyn, it was my privilege to make a report (Journal of the A. M. A., February 4, 1922) in which listed among the conclusions will be found the following: "According to our experience to date, butyn in the quantity required is less toxic than cocain."

In beginning the use of butyn in an experimental way, the committee was confronted with a statement of the Research Committee of the Council on Pharmacy and Chemistry of the A. M. A., to the effect that butyn is two and one-half times more toxic than cocain when injected hypodermatically into albino rats, and that the lethal dose of butyn, when injected intravenously into cats, is about equal to that of cocain. One member of our committee conducted some animal experiments which confirmed those of the Research Committee concerning the toxicity of butyn. Therefore, the committee began the clinical use of butyn with great caution, and it was only after butyn had been used in hundreds of cases by the various members of the committee for minor and major operations on the eye, as well as in operative work in the nose and throat, and without noting the slightest systemic toxic manifestations, that the committee felt justified in pronouncing butyn relatively safe. We were not unmindful, and so stated in the report, of the possibility of there being frequently discovered idiosyncrasies in the human with the development of toxic effects, though we were not perpared after such extended clinical experience on our part, and on the part of many others in various parts of the United States, to expect fatal results coming on with tragic suddenness and apparently with no chance of relief. Unfortunately, I, the chairman of this committee, and for over two years having a relatively large experience in the use of butyn as a local anesthetic, have had two fatalities within a week, and it is but a duty that these should be reported in the interests of scientific advancement, and safety of operative patients who are to undergo operations under local anesthesia. The two fatal cases occurring probably as a direct result of the toxic effects of butyn are as follows:

CASE 1. Mrs. H., age 31; married; mother of one child; no miscarriages; family history negative. Patient gave a history of obstructed breathing and discharge from both sides of the nose dating back several years. Had frequent head colds accompanied by some coughing, and almost constant morning headache. The family physician reported urinalysis and general physical examination negative. The nose presented a marked deviation of the nasal septum to the right, with a bony ledge extending clear through. The upper portions of both nasal passages were filled with polypi, and on the left side the tissues were bathed with mucopurulent secretion. Resection of the nasal septum, to be followed by removal of the polyps and exenteration of the anterior ethmoidal cells, was advised and accepted. Patient given

morphin 1/4 grain, atropin 1/150 grain hypodermically. Nasal chambers washed with sterile saline solution, after which each side of the septum was swabbed with a pledget of cotton saturated in five per cent butyn solution, to which had been added a few drops of 1/1000 adrenalin solution. A few minutes later, this was repeated, followed immediately by the application of flat pledgets of cotton moistened with the same solution, considerable care being exercised to have these moist pledgets applied against the mucous membrane. These pledgets, from which the surplus butyn solution was squeezed, were permitted to remain in contact with the mucous membrane of the nose about ten minutes, according to the usual custom, when they were all removed carefully with forceps. Throughout the time required for this anesthetization, the patient was in a reclining position on the operating table and prepared for the operation. After removal of the pledgets, and while sponging the nasal passages prior to the beginning of the contemplated operative work, the patient suddenly, and without the slightest warning, went into a violent convulsion. Almost at the very minute when the convulsion began, the patient had been laughing and talking, and the trained nurse who, according to usual custom, carefully watches the condition of all patients undergoing operative work, had her finger on the radial pulse and reported that the pulse was regular and of good quality.

At the onset of the convulsion, the patient's head was lowered and a heart stimulant consisting of digitalin 1/100 grain, nitroglycerin 1/100 grain, strychnin sulphat 1/30 grain, atropin sulphat 1/250 grain, was administered hypodermically. The patient recovered from the first convulsion and was regaining normal respiration and normal heart action, when she went into another convulsion. Adrenalin was then injected directly into the heart muscle. One or two more convulsions occurred, of less violence, and the patient ceased to breathe and all efforts at resuscitation failed. The convulsions followed one another rapidly, but during the intervals the heart was beating in a fairly normal way and sufficiently to give a good radial pulse. During the interval between the first and second convulsions, an inhalation of ether was tried, and following the final convulsion artificial respiration was introduced. Autopsy was requested but not permitted.

CASE 2. Mr. K., student, age 15. Family history negative; general health good. Gave a history of a fall on the face eight months previously, resulting in obstructed breathing on the left side. Examination disclosed very marked deviation of the nasal septum to the left, with a sharp angular spur near the floor

anteriorly. Submucous resection of the septum advised and accepted. Patient given morphin 1/6 grain, atropin 1/150 hypodermically, followed by cleansing the nasal passages with a sterile saline solution. Subsequently, the patient was placed on the operating table in a semireclining position, and the nasal septum swabbed with a five per cent solution of butyn, to which a few drops of adrenalin had been added. This was followed by the careful placing of flat pledgets of cotton moist with the five per cent butyn-adrenalin solution against the septal mucous membrane, all accessible portions being covered. These pledgets had been in the nose perhaps eight or ten minutes, when one of the two nurses who were in the operating room caring for the patient and preparing the instruments for the operation called me hastily from an adjoining room, and I found the patient in a violent convulsion. A heart stimulant consisting of digitalin 1/100 grain, nitroglycerin 1/100 grain, strychnin sulphat 1/30 grain, and atropin sulphat 1/250 grain, was administered hypodermatically, and a cone saturated with ether placed over the nose. After an interval of a few seconds, the patient went into a second convulsion, when adrenalin was injected directly into the heart. He stopped breathing at the end of a third convulsion, and all efforts at resuscitation, including direct manipulation of the heart through an incision made for the purpose, were unavailing. The nurses declare that the heart action was excellent right up to the time of the convulsion, and the patient was in good mental spirits.

An autopsy was permitted and resulted in finding nothing of significance except evidence of a disturbed endocrine system. At the autopsy, information was volunteered that the father had an idiosyncrasy for a local anesthetic, probably cocain, but so far as known, there is no other local anesthetic idiosyncrasy in the family.

In these two cases, everything points to the toxic effects of the local anesthetic as being the cause of the deaths. The solutions used were identical in strength to those regularly used in operative work in the nose, prepared in the usual way, and the butyn was taken from a bottle from which other solutions had been made and had been used without ill effects. In fact, during the interim between the dates of the two deaths, several major intranasal operations were uneventfully performed under local anesthesia, produced by use of solutions made from butyn from the same bottle from which the solutions used in the fatal cases were made. The two deaths occurred a week apart, and prior

to these deaths I am satisfied that butyn in five per cent solution, to which a few drops of adrenalin had been added, had been used in a good many hundred operative cases, including many submucous resections of the septum and operations upon the sinuses, in which as much if not more of the anesthetic had been employed, and without the slightest suspicion of toxic symptoms. Because of the large experience with butyn without seeing any toxic effects, the first fatality was not considered to be due to the anesthetic, although the possibilities were discussed.

In the first case, there was an abundance of pathology to justify the belief that an intracranial catastrophe not associated with the anesthetic could be responsible for the death, but failure to secure an autopsy did not enable us to prove or disprove the assumption. The second fatality occurred in an apparently healthy boy, with a deformity within the nose as the only reason for the operation. The autopsy in this case was attended by the president, the chemist and the pharmacologist of the Abbott Laboratories, manufacturers of butyn, and it was not until their visit that I became acquainted with their investigations in an effort to find an antidote for the toxic effects of butyn, as well as for cocain and other local anesthetics. They informed me, and the results of their investigations have been published (*Journal of Laboratory and Clinical Medicine*, April 1923), that in animals the toxic effects of butyn have been counteracted successfully by the administration of pituitary solution, and that the favorable results from the treatment are noted when animals are given what is known to be a fatal dose of butyn. In fact, they report that even after the onset of convulsions the administration of pituitary solution brought about recovery. During their experiments, a considerable number of drugs were investigated for their detoxicating action, including epinephrin, caffen citrat, hyoscin hydrobromid, morphin sulphat, atropin sulphat, hydrastinin and pituitary solution. The latter appeared to be superior to the other drugs investigated, and they believe that it holds out promise both as a preventative of undesirable symptoms from sublethal doses as well as an antidote in poisoning by fatal doses of any of the local anesthetics, butyn included. Judging from their experiments, small therapeutic subcutaneous doses of pituitary solution, injected simultaneously with a local anesthetic, are sufficient to eliminate the fall in blood pressure from therapeutic doses, as well as the convulsions from higher doses and death from fatal doses. Used as an antidote in cases where the convulsions already have set in, pituitary was effective only when

administered intravenously. Undoubtedly it deserves clinical trial. Obviously, it should not be used in pregnant or in high blood pressure patients.

It should be borne in mind that while butyn may be relatively safe in a large proportion of cases, yet evidence seems to show that even in small doses it is highly if not fatally toxic to certain individuals, and the fatal symptoms come on so rapidly and with so little warning as to make it highly questionable whether pituitrin solution or any other supposed antidote will prove efficacious. I am not unmindful of the fact that cocain, as ordinarily used, is fatal in some patients, and perhaps, as already suggested, the idiosyncrasy for cocain is even greater than it is for butyn, but in cocain poisoning you almost invariably have some warning, whereas in butyn the fatal symptoms seem to come on with such suddenness as to preclude the possibility of saving the patient's life. I, therefore, feel that in view of my unfortunate experiences, following on the heels of a long period of favorable results with butyn as a local anesthetic in many hundreds of operative cases, I am justified in urging my confreres that if they use butyn, it should be with the utmost caution and with everything at hand to counteract toxic symptoms should such develop. This opinion is all the more justified, in view of previous published endorsement of butyn as a trustworthy local anesthetic after having used it extensively for many months.

DISCUSSION.

DR. EMIL MAYER, New York City: The final meeting of the General Committee of the American Medical Association on the toxicities following local anesthesia was held a week ago. Forty-one deaths had been reported and the details obtained. This Committee, representing some of the largest hospitals and teaching institutions of this country, made an analysis of these, a study of the symptoms in toxic cases, methods used to resuscitate, as well as an inquiry into causes of death as disclosed by twenty necropsies. Following this, the Committee has agreed upon recommendations which, if followed, would in its opinion prevent many and lessen the number of fatalities. This report, when completed, will be sent to the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association. When approved it will undoubtedly be published in the Journal of the American Medical Association.

The brief time at my disposal in this discussion will not permit me to go into the general matter of fatalities. My remarks will be confined to the subject of the paper. On behalf of the Committee, I desire to express its sincerest commendation of Dr. Bulson's rare and splendid courage in presenting the full histories of his fatalities, solely for the benefit of the profession, and because he believes that justice demands a truthful statement.

His action is one to be emulated, for there are not many who possess the courage of their convictions, as has the reader of this paper. He has conferred a lasting service. None can appreciate this more than myself. Four years' work on this question has taught many things. Doctors fail to answer ordinary letters sent them on matters of vital interest to themselves to a surprising extent. Often when asked as to fatalities that they are alleged to have witnessed, we have been met by denials, misstatements or the silence of the grave.

In my official capacity, I learned from nonconfidential sources of a death following the use of butyn, which was alleged to have occurred in the practice of Dr. H. R. T. Ross of Ladysmith, Wisconsin. Letters to him asking for confidential information as to details, explaining the great importance that such information would be to my Committee, and appealing to the recipient's sense of duty to the profession and humanity, and as a member of the American Medical Association; letters sent to him by one of the professors of his own state university, as also a letter sent to the physician who was registered as Secretary of the Rusk County Medical Society, have been simply ignored.

You can realize what this means, when I state that the deaths from this new drug are the first to be recorded, and we all want to know in fairness to this drug whether it alone is responsible, whether other toxic drugs were used, what the strength was, and how applied. Like every new drug, butyn is still on trial. It has been most highly endorsed, and it may readily be possible that our dosage has been too high, or the combinations with other drugs harmful, and when these are known, it will receive its proper place in our armamentarium. To avoid any misconception, I wish to state that my Committee is in no sense opposed to local anesthetics, and that we hold no brief for any of them.

During the four years of my Chairmanship of investigating committees on toxicities following local anesthesia, I have conferred frequently with the representatives of the manufacturers of these anesthetics, and have met nothing but the heartiest cooperation. They even went so far as to give me the facts in their possession of fatalities following the use of the drug they manufacture. None has been more frank and straightforward than the manufacturers of butyn, a most commendable act, which I am sure the profession will be grateful for.

Finally we appreciate what it means to the practitioner who is unfortunate enough to have had a fatality, for whom we have the sincerest sympathy. In a scientific presentation, we cannot show this or what it means to him. Having assured the patient of the painless, bloodless and simple nature of the operation, he employs (or thinks he does) the methods he has always used. Within an incredibly short time, his patient becomes ghastly, faints, convulsions occur, help is hastily summoned, all possible restoratives used and rapidly applied, to no avail. The unsuspecting happy patient of a few minutes ago has ended his earthly career, and a valuable life is lost.

Shocked beyond measure, compelled to condole with and explain to survivors, the surgeon faces publicity, front page headlines, interviews with reporters and the coroner, bitter accusations. His books show few references to mortalities; his journals still fewer. Nobody has warned

him of the danger, or told him what to use and be prepared for, hence when a committee to study his and similar cases asks him to state what really happened, he is resentful; he wants to forget it; he becomes irritable, says nobody gave him any information, so he will give none, and consigns his inquirers to everlasting torment.

He fails to realize that the gathering of cases similar to his own, studied by men accustomed to analysis, will be of actual help, not only for his future guidance and that of others, but also a protection in a possible suit for damages.

DR. JAMES J. KING, New York City: I wish to commend the courage of Doctor Bulson in bringing this very interesting and important report to us. I wish to call attention to two points.

Experiments on animals prove that butyn is more toxic than cocain, and in view of this fact, it should be used with the greatest caution until we know more about it. This is true of all local anesthetics. If the experimentation on animals proves that it is highly toxic, it will likely be highly toxic for humans, and no matter what your clinical experience over a short period of time may be, we should rely upon the results obtained in animal experimentation. That was shown in the early experiments with animals and butyn.

Another point which has been fairly proven lately, is that most local anesthetics are more toxic for animals when they are combined with adrenalin than without adrenalin.

DR. ALBERT E. BULSON, Ft. Wayne, Ind. (closing): I am aware of the fact that there is a justifiable hesitancy on the part of nearly all medical men to report their fatalities. The committee of which Dr. Mayer is chairman knows that there are many fatalities caused directly from the use of local anesthetics, the reports of which have never been published. Unless we have the courage to report fatalities, we are not doing full justice to the medical profession and to our patients, who place confidence in us. We should place duty to the profession and to humanity above any personal feelings.

Another reason for reporting these cases, is because I had tentatively reported favorably upon the use of butyn, in a report to the Council on Pharmacy and Chemistry of the American Medical Association concerning the clinical use of butyn. I have used butyn, in the past, in perhaps hundreds of cases in which a major operation within the nasal cavity was performed, and with the greatest success and satisfaction. Therefore, it was a great surprise to me to have two fatalities after such extended use of the drug. No doubt, if I had used butyn for my ophthalmologic work alone—for the quantity of the drug used is always less than that used in rhinologic work—I might not have discovered any toxic effects. However, I am not unmindful of the fact that an idiosyncrasy to butyn may produce, even when used in the eye alone, serious if not fatal results.

Concerning the results of experimentation, it should be noted that the higher the development of animals, the less toxic the effects from butyn. Thus if butyn is injected into chimpanzees and apes, the toxic effects are less marked than when injected into albino rats. Reasoning by analogy, you would expect to have less toxic effects in the human than in the lower animals. There is also another phase of the subject to be

considered, and that is the selective action, for it is quite possible that animals have idiosyncrasies for drugs exactly the same as have human beings. Therefore, it will take more than a series of experiments on a few animals to prove or disprove the toxicity of any local anesthetic when used on the human.

Finally, I wish to have it understood that I am not discouraging the cautious use of butyn, but I am unhesitatingly condemning the use of butyn without due regard for its possible toxic and even fatal action, even when employed in what is considered relatively safe doses.

TREATMENT OF OPTIC NERVE INVOLVEMENTS AS DETERMINED BY OPTIC CANAL RADIOGRAPHS.

LEON E. WHITE, M.D.

BOSTON, MASS.

In a recent anatomic study at the Harvard Medical School, the optic canals were found to vary in size from 4 to 6.5 mm., and in shape from the usually circular to various degrees of ovalness; so the question naturally arose, as to whether or not such variations would explain why some nerves become involved and others escape.

It was also found that the smaller the canal the more extensive the pneumatization around it. Therefore, it seemed that there would be greater likelihood of nerve involvement in a relatively small, pneumatized canal, than in a larger one less extensively pneumatized. The mucosa of the paranasal sinuses, when infected, undoubtedly offers to the more extensively pneumatized canal a larger area for infection to reach the optic nerve. The bony walls of these excessively pneumatized canals were also found to be thinner, thus leaving but a slight barrier to infection. It seems logical to conclude, that when infection invades one of these small osseous canals, it requires but moderate swelling to produce a constriction so severe that nerve degeneration would ensue. The optic canal is usually circular; a few are flattened on top, and in these the pneumatization is pronounced in the lesser wing above the canal.

When the canal is oval (a rather rare abnormality), it means pneumatization both above and below it. If triangular (a very infrequent shape), there is an extension also of the pneumatization into the bridge formed by the lateral root of the lesser wing of the sphenoid. Any irregularity in the contour of the canal is usually associated with a diminution in its caliber. Canals under 4.5 mm. are, as a rule, irregular, yet enough circular ones are found to demonstrate that the shape is not always an indication of the size.

The conclusions from this anatomic study are, that both the shape and size of the canals are dependent largely upon the amount of pneumatization around them.

The next step to determine is whether the canals are below normal in patients with optic nerve disturbances. Fortunately, it was possible to have the canals filmed in twenty-five such cases,

and for comparison a similar number was made of normal individuals. The canals were then carefully measured and the results tabulated.

Heads differ so much in contour, that it is extremely difficult to obtain a position where the correct shape and size of the canal will appear on the film; in fact, the first canals all appeared small and oval. Later, it was discovered that if the skulls were held up to the light, so that the full diameter of the canal could be seen, the line of vision was always somewhere through the lower outer quadrant of the orbit. When viewed from the other quadrants, the canal usually appears narrowed or flattened. This did not become apparent until a number of cases were filmed, so that much of the work had to be repeated. It is not always possible, even now, to get the image of the canal in the lower outer quadrant on the first or even second attempt. The position as worked out by Doctor A. S. Macmillan, is as follows: The patient lies with one eye at a time over the plate, the nose and the malar bone touching it, while the forehead is about a finger's breadth away. The ray penetrates from the region of the posterior parietal protuberance of the opposite side.

To establish the normal size and shape of the canals, twenty-five doctors and attendants from the Infirmary volunteered as subjects, and fifty fairly good radiographs were obtained. Of the fifty radiograms in this normal series, five were slightly oval, and the average diameter was 5.35 mm. The average diameter of the canals in the twenty-five cases of optic nerve involvements was 4.68 mm. Twenty-six of these canals were oval. This makes practically 50% oval as against 10% in the normal series. There was about $1/2$ mm. difference between the vertical and horizontal diameters in the optic nerve series, as contrasted with about $1/3$ mm. in the normal series. There was $2/3$ mm. difference between the canals in the normal and optic nerve cases. In nineteen of these twenty-five oval canals, the optic nerve was involved. Of the twenty-four round canals, there was involvement of the nerve in twelve. In the nineteen oval canals with optic nerve involvement, five were bilateral. Twelve were operated upon; one recovered without operation; and in the other (unoperated upon) there was no improvement. Of the ten patients with round canals, one recovered without operation, six had intranasal operations, and in three the tonsils were removed.

Large round canals are frequently found in both acute and

chronic cases. In these, it is thought the neuritis is due to bacteremia or toxemia—usually teeth or tonsils—rather than to direct extension from infection in the sinuses. Large canals, let me interpolate, generally contraindicate accessory sinus surgery.

Is there any relationship between the size of the canals and the severity of the neuritis; and if so, what bearing should this relationship have on treatment? The data from the following cases will, I believe, answer these queries.

Thirty-six cases with optic nerve disturbance are cited in chronologic order, briefly stating the duration, vision, size of canals, method of handling and results. The cuts illustrate some of these canals. It may be of interest to note that all these cases, the earlier ones dating back nearly ten years, have been checked up at frequent intervals.

CASE 2. Bilateral. 10 days' duration. Vision 10/100 left, with some blurring in right. Fundi negative. Optic canals 5 mm. Middle turbinate removed and sphenoid opened. Improvement rapid. Normal vision in two weeks. This patient would probably have recovered under local treatment, as the canals were practically normal, but at that time we were unable to determine this. This case illustrates the type where one may with safety delay surgical intervention.

CASE 4. Bilateral. Duration six weeks. Vision 2/200. Marked optic atrophy. Canals 4 x 5 mm., i.e., oval. Both middle turbinates removed and sphenoids opened. No further loss of vision; slight improvement. Earlier operation would probably have been of greater benefit.

CASE 6. Unilateral. 5 days. Vision nil. Blurring of edges of disc. The canal on the unaffected side was 5 mm., affected side 4.5 mm. Septum resected, middle turbinate removed and sphenoid opened. Ultimate vision 20/20. This case, with a rather small optic canal, indicated operation. The process was unusually acute, and there would probably have been marked impairment of vision had not active surgical treatment been instituted.

CASE 7. Bilateral. Duration three weeks. Vision light perception. Canals 4.5 x 5 mm. Marked neuritis with blurring of edges of discs. Usual intranasal operation, i.e., the removal of the middle turbinate, opening of sphenoid and posterior ethmoid cell. Result, vision 20/30, with some pallor of the nerve.

CASE 8. Bilateral. Duration six weeks. Vision light

perception right, fingers three feet left. Canals 4 x 4.5 mm. Blurring of edges of discs, and marked neuritis right. Removal of middle turbinate, opening of right sphenoid and resection of septum. Normal vision. This case with its small oval canals is the type which seems to be greatly benefited by operation.

CASE 9. Both eyes affected but not simultaneously. Duration right, five weeks; left, one week. Vision 20/200 right, 20/40 left. Marked engorgement of fundi. Canals 4 x 5 mm. Usual intranasal operation on both sides and left antrum opened. Vision normal. This case, with oval canals considerably below the normal, seemed greatly benefited by the operations.

CASE 13. Bilateral. Duration four months. Vision 20/200. Progressive loss. Temporal pallor. Canals 3.5 x 4 mm. Usual intranasal operation. Vision 20/100. The smallness of these canals was probably a factor in the loss of vision and had there been a more acute process, much more destruction would have resulted. The operation was done to prevent further impairment, and in this it succeeded.

CASE 23. Unilateral. Duration seven days. Vision nil. Edges of disc slightly indistinct. Canals 4.5 x 5 mm. right and 5 x 5.5 mm. left. Usual intranasal operation. Ultimate vision 20/20. The vision returned slowly, and there was some pallor of the nerve. The affected eye was (as is usual) on the side of the smaller canal.

CASE 24. Unilateral. Eleven days' duration. Vision nil. Disc hazy and indistinct. Canals 5 mm. Usual intranasal operation. Fingers at two feet. Marked optic atrophy. The operation was of but slight benefit, even though done within eleven days. This case is unique, in that over three years after the loss of the vision in the right eye the left eye became affected, although not to the same extent. There was nothing in the nose to suggest operation, especially as the optic canals were practically normal. The tonsils, which showed evidence of infection, were removed. Little, if any, benefit resulted. The case was thoroughly investigated, but nothing in her physical, neurologic or serologic examination offered any explanation for the loss of vision.

CASE 25. Unilateral. Nine days' duration. Light perception. Disc somewhat blurred. Canals 4.5 mm. Usual intranasal operation. Normal vision with some pallor of the nerve. In such a severe neuritis associated with small canals,

the operation was, I believe, of marked benefit. Had it been delayed, the resulting slight pallor would undoubtedly have been greater. The determination of the size of the canals greatly assists one in deciding between operative procedure and palliative treatment.

CASE 26. Unilateral. Duration five days. Vision fingers one foot; fundus normal. Canals markedly oval, 4×5.5 mm. Usual operation. Vision 20/40, with some temporal pallor. The oval canals probably indicated excessive pneumatization, but one might have treated such a case had the size of the canals been known.

CASE 27. Bilateral. Three and one-half years' duration. Vision nil. Complete optic atrophy. Canals 3.5×4 mm. No treatment. Permanent and complete blindness. These canals were the smallest measured, and slightly oval. It would have been of inestimable value at that time had the importance of filming the canals been comprehended. Practically all my data had been accumulated before this patient was seen, and when referring her to Doctor Macmillan, the radiologist, I remarked that if her canals were not the smallest of any filmed, I would scrap my winter's work on this subject. It was gratifying to learn that they were both extremely small and slightly oval. This case, more than any other, has led me to believe that it is necessary wherever there is a neuritis as marked as it was in this child, to ventilate the posterior sinuses promptly, unless some other definite cause can be discovered. I did not see her until some years after she became blind, but was able to obtain a very complete history (see previous report of case), and believe that with our present knowledge, some of her vision at least might have been saved.

CASE 28. Bilateral. Recurrent. Vision 20/200. Fundi normal. Canals 4×5.5 mm. Local treatment. Vision 20/30—. The poor general health and the rapid improvement under treatment contraindicated intranasal surgery. The canals were markedly oval, there being 1.5 mm. difference between the vertical and horizontal diameters. The area of the canals was but slightly below normal. There have been occasional slight recurrences. This case is of interest showing that the nerves are susceptible to disturbances in the nose, the unusually marked ovalness indicating extensive pneumatization. The size of the canals, however, favor recovery.

CASE 31. Unilateral. Three days' duration. Fingers

three feet. Edges of disc practically obliterated. Small blood vessels engorged. Canals 5 mm. Usual intranasal operation. Vision 20/20, with some temporal pallor. This case was unusually acute, and although the canals were of nearly normal size, operation seemed to be indicated. Had it been possible at that time to determine the size of the canals, one might have waited longer.

CASE 33. Unilateral. Duration three weeks. Vision fingers six feet. Temporal pallor of disc. Canal on affected side 4 x 5 mm.; on the unaffected side 4.5 x 5.5 mm. Usual intranasal operation. Ultimate vision 20/30—. As is usual, the affected side was the one with the smaller canal. The ovalness of the canals was also pronounced.

CASE 34. Unilateral. Ten days' duration. Vision, shadows. Edges of disc indistinct. Canals 4.5 mm. Removal of middle turbinate and tonsils. Vision 20/30.

CASE 36. Unilateral. Duration ten days. Light perception. Edges of disc blurred. Canals 4 x 4.5 mm. Usual intranasal operation. Vision 20/30, with temporal pallor. The canal of the other eye was one mm. larger in both vertical and horizontal diameters. This case again seems to demonstrate the susceptibility of a small canal, and the necessity for ventilation of the posterior sinuses.

CASE 38. Unilateral. Duration three weeks. Vision 20/100. Blurring of edges of disc and choroiditis. Canals 5 mm. Local treatment. Normal vision. The filming of the canals in this case gave assurance that recovery could be expected without operation. This is the type of case that usually recovers spontaneously.

CASE 41. Unilateral. Duration two weeks. Vision 2/200. Canal 4.5 x 5 mm. Fundus normal. Local treatment. Vision 20/30—. The canal of the unaffected side was round and one-half a millimeter larger. The filming of this canal led me to expect recovery, even though the canal was somewhat below normal size. The age of this patient (67) also rather contra-indicated operation. Here again the neuritis was on the side with the smaller canal.

CASE 42. Bilateral. Ten weeks' duration. Vision fingers at seven feet. Fundi negative. Canals 4 x 5 mm. Refused operation. But slight temporary improvement under local treatment, i.e., fingers at thirteen feet. Six months later,

vision dropped again to seven feet. These small oval canals were probably a factor in the loss of vision, and I believe that had the patient consented to the usual intranasal operation, he would have been benefited.

CASE 44. Unilateral. Two months' duration. Vision 12/20. Fundus normal. Canals 5 mm. Local treatment (tonsils removed later). Normal vision. This mild case, with normal canals, contraindicated any intranasal surgery.

CASE 45. Unilateral. Two weeks' duration. Vision nil. Disc edges blurred, definite papillitis. Canal 4 x 5 mm. Intranasal treatment. Diseased tonsils removed. Vision 20/30, with some temporal pallor. There was no marked blocking of the posterior sinuses. The neuritis, as usual, was on the side of the smaller canal. The canal of the other side was round and 5 mm. in diameter.

CASE 46. Bilateral. Five years' duration. Recurrent. Vision 20/200. Partial optic atrophy. Canals 5 mm. Resection of septum and removal of diseased tonsils. Vision 20/100. The normal canals seemed to indicate toxemia rather than any direct accessory sinus infection. The removal of the diseased tonsils, it is hoped, will eliminate the focus responsible for the recurrent attacks.

CASE 47. Unilateral. Duration four days. Fingers at four feet. Fundus normal. Canal 4.5 mm. Diseased tonsils removed. Normal vision. The diameter of the canal on the affected side was 1 mm. less than that of the other side. There was no evidence of blocking of the accessory sinuses, so they were not disturbed. Here again the neuritis was on the side of the smaller canal.

CASE 48. Bilateral. Duration ten years. Vision 10/200. Optic atrophy. Canals 5.5 mm. Removed diseased teeth and tonsils. The accessory sinuses appeared normal. This seemed to be a case where the marked infection in the teeth produced a toxic neuritis.

CASE 49. Unilateral. Duration six weeks. Vision 20/70. Edges of disc blurred. Canal 5 mm. Septum resected, middle turbinate removed. Vision 15/30—. Although this case was improving when operated upon, there was so much blocking by the middle turbinate and septum that these needed correction, irrespective of the neuritis. The tonsils likewise were diseased. Had there been no definite focus of infection, this case with practically normal canals would not have been operated upon.

CASE 50. Bilateral. Duration nine months, progressive. Light perception in one eye, 20/70 in the other. Discs show some temporal pallor. Canals 5 mm. Usual intranasal operation on both sides and removal of tonsils. Vision unimproved. As this and the following cases have not been previously reported, a somewhat more detailed description seems indicated. W. A., 33, was referred from the Eye Clinic on April 6, 1923, with diagnosis of bilateral retrobulbar neuritis. The vision in the right eye had been failing for nine months, and in the left eye six months. She was referred by Doctor Clay of Atlanta, Ga., to Doctor Harvey Cushing, on the supposition that the loss of vision might be due to a pituitary tumor. After investigating her, Doctor Cushing wrote me, "I do not believe she has a brain tumor though it is conceivable that there may be a suprasella growth which has given bizarre symptoms". This case was not typical of either accessory sinus or pituitary blindness. She was very anxious to take every possible chance for relief, so that in spite of the fact that the optic canals were practically of normal size, the usual intranasal work was done. It is true that there was considerable blocking of the nose by hypertrophied middle turbinates, and the tonsils showed some evidence of disease. The patient's health two months after operation was reported better, and the "dreadful headaches" gone. The normal size of the canals bears out the belief, previously expressed, that in such cases accessory sinus surgery is valueless as far as the relief of the amblyopia is concerned. Since this writing, I have learned that the vision in the better eye had recently failed rapidly, and that on September 21, 1923, a suprasella tumor was removed by Doctor Dandy of the Johns Hopkins Hospital. This bears out the previously expressed opinion that the loss of vision was in no way associated with the accessory sinuses.

CASE 51. Bilateral. Duration nine months, progressive. Light perception in one eye, 15/60 in the other, slight temporal pallor. Canals 6 mm. (the largest with one exception in the series). Usual intranasal operation on both sides, resection of the septum and removal of tonsils. Vision continued to fail, and in three months was 4/200. This case, W. A. W., 32, was referred by Doctor Harvey Cushing on April 6, 1923, with the statement that he found nothing in the way of a brain tumor to account for the loss of vision. The patient was in good general health and had been carefully investigated, but nothing

was discovered to account for the gradual loss of vision. This had commenced to fail in the right eye nine months previously, and when seen there was only light perception. The vision in the other eye commenced to fail two months ago. There was a marked deflection of the septum, with blocking of the posterior sinuses by enlarged and degenerated middle turbinates. The tonsils showed some evidence of infection. This case, like the preceding one, did not offer much hope of improvement, but was of such a nature (progressive loss of vision) that an attempt at relief seemed advisable. No benefit resulted from the opening of the sinuses, and not even the progressive loss of vision was checked, as it seemed to be in the preceding case. These two cases (50-51) convinced me that large canals contraindicate intranasal surgery.

CASE 52. Unilateral. Duration two weeks. Vision 20/100—. Canals, 5 mm. Fundus normal. Abscessed tooth extracted. Local treatment. Normal vision. A.E.S., 35, was referred by Doctor Cheney on May 8, 1923, with diagnosis of retrobulbar neuritis, left. The septum was deflected to the opposite side, with compensatory hypertrophy of the left middle turbinate. On filming the teeth, there was found to be an area of rarefaction about one of them, and on its removal it was found to be badly diseased. It was felt that with normal canals, the case should recover without surgical intervention, as it promptly did, with normal vision, in six weeks.

CASE 53. Unilateral. Duration two weeks. Vision 20/200—. Fundus normal. Local treatment. Vision 20/20. D.C., 23, referred from the Eye Clinic on May 17, 1923, with diagnosis of retrobulbar neuritis, right. Although the canals were rather small, the neuritis was mild and the case progressed so favorably, that there was no indication for intranasal surgery. There was no special blocking of the nose, and the tonsils had already been removed.

CASE 54. Bilateral. One eye many years, the other of six weeks' duration. Vision fingers at two feet. Discs pale. Canals 5.5 mm. Abscessed teeth extracted. Vision in recently affected eye 20/50—, the other unimproved. E.DuB., 52, referred from the Eye Clinic with diagnosis of optic atrophy, right; retrobulbar neuritis, left. The canals being of normal size, the sinuses were not ventilated. Diseased teeth were probably the source of the neuritis, and prompt improvement followed their removal.

CASE 55. Unilateral. Duration, three months. Vision 20/40. Fundus normal. Canals 4.5 mm. Resection of the septum and

removal of abscessed teeth. Vision normal. E.T., 30, referred from the Eye Clinic on May 4, 1923, with diagnosis of retrobulbar neuritis. The neuritis was rather mild, and although the canal was somewhat below normal, there seemed no indication for opening the sinuses. The septum was obstructive and needed correction. The operation seemed to be of distinct benefit.

CASE 56. Unilateral. Duration three weeks. Vision 20/60 Fundus normal. Canals 6×5 mm. Local treatment. Normal vision. G. N. B., 37, referred by Doctors Cheney and Crockett on May 31, 1923, with questionable diagnosis of retrobulbar neuritis, right. Pyorrheal pockets and abscessed teeth were discovered in radiographic examination, and recovery promptly followed the elimination of these foci. This case leads one to conclude, that with a normal canal, the source of the infection is probably not in the accessory sinuses. The peculiar point in this case is, that the vertical diameter of both canals was greater than the horizontal, the only one in the series showing this abnormality. Another peculiar feature was that the left (the unaffected side) was smaller than the right. The general statement that the smaller canal is always the one affected holds true only when the canals are below the normal size.

CASE 57. Unilateral. Recurrent for some years. Vision 20/40—. Fundus negative. Canals 4.5×5 mm. Removal of abscessed teeth. Vision unchanged. C.R.W., 50, referred by Doctor Hatch on June 8, 1923, with diagnosis of recurrent retrobulbar neuritis, left. This patient had had occasional periods of blurring of vision for some years, with gradual loss. There seemed no evidence of obstruction to the sinuses. Abscessed teeth were found, and it is hoped their removal will prevent recurrence. Time enough has not yet elapsed to know the final outcome.

CASE 58. Bilateral. Duration six months. Vision 4/200. Fundi show temporal pallor. Canals 5×5.5 mm. Tonsils removed. Vision 20/60 right, fingers (4 feet) left. C. N., 19, was referred by Doctor Verhoeff from the Eye Clinic on June 13, 1923, with diagnosis of retrobulbar neuritis. The physical and neurologic examinations were negative. There was some question of wood alcohol or arsenic poisoning. The tonsils were diseased, with enlargement of the cervical glands. Improvement followed their removal. The neuritis, as one would expect from the normal size of the canals, was probably toxic. There was no evidence warranting the opening of the accessory sinuses.

CASE 59. Unilateral. Duration two years. Vision nil. Disc white. Canal 6×6.5 mm. Removal of diseased teeth. No improvement. H. L., 50, was referred from the Eye Clinic by Doctor Hatch on July 17, 1923, with diagnosis of optic atrophy, right. This case was for diagnosis, as nothing could be expected in the line of treatment, there being complete atrophy of the nerve. The blindness came on within four weeks, two years ago, and has never changed. The optic canal of the affected side was the largest in this series, irregular in outline and apparently deficient in the lower outer quadrant. It seems probable that something (possibly some benign growth) produced enough pressure on the nerve at this point to produce atrophy. The canals in case 51, which were also abnormally large, suggested some new growth, as there was some irregularity and deficiency in their contour.

SUMMARY OF THE FINDINGS IN 36 CASES OF OPTIC NERVE INVOLVEMENT.

In ten cases, the vertical diameter was 4 mm. or less, and all were oval. Two of these were benefited but slightly, if at all, by operation. One unoperated case had complete optic atrophy. Another refused operation and was unimproved. Four cases improved greatly after the opening of the posterior sinuses, but there remained marked temporal pallor. One required a tonsil operation, and the remaining case, unoperated upon because of poor general health, has had frequent recurrences. These were, by far, the most serious cases in the series.

There were ten cases with canals approximately 4.5 mm. in the vertical diameter, and of these but three were oval. Four had the posterior sinuses opened, and all recovered with practically normal vision. Five had teeth or tonsils removed, and all these made good recovery. The remaining case recovered under local treatment. It is interesting to note that in these 4.5 mm. canal cases, every one recovered with practically normal vision.

There were eleven cases with optic canals 5 mm. in the vertical diameter, and of these but two were slightly oval. In three *chronic* cases where the etiology was in doubt (the neuritis being from some months to several years in duration), one showed marked improvement after removing the tonsils. A septum and tonsil operation on the second was followed by slight improvement. The third case, with progressive blindness, was temporarily improved in health and relieved of severe headaches, but later had a brain tumor removed. Four *acute* cases had the posterior sinuses opened; normal vision in three, and but slight improve-

ment in the fourth. The neuritis in this case was apparently not of nasal or dental origin. Three *acute* cases recovered under local treatment. The remaining case made a good recovery following the removal of tonsils and middle turbinate. Of the eight acute cases, seven recovered with normal vision, and in the obscure eighth case, tabes or multiple sclerosis is suspected.

There was one case with canals 5.5 mm. in diameter. In one eye there was optic atrophy of many years' duration; the other eye, of six weeks' standing, improved rapidly after the removal of abscessed teeth.

There were three cases with canals abnormally large, i.e., of 6 mm. One of these, a case with progressive loss of vision, continued to fail after the sinuses were opened and the tonsils removed. The neuritis was probably not of nasal or tonsillar origin, possibly pituitary.

In the second case, the vision returned to normal after the removal of the foci of infection in the mouth. The third case, untreated, had complete optic atrophy of two years' duration, probably from abscessed teeth.

Further analysis of this data reveals, that in the ten cases with canals of 4 mm. or less, diseased teeth were extracted in one, and in another the tonsils were removed. Of the ten cases with canals of 4.5 mm., five had either infected teeth or tonsils, and the removal of these undoubtedly played an important part in the recovery. Thirteen of the fifteen cases with canals of 5 mm. or over had teeth or tonsils removed. These figures greatly surprised me as I finished tabulating them, although I had felt for some time that large canals were not usually associated with cases requiring operations on the accessory sinuses.

The optic canals of the same individual were occasionally found to differ in size. In the thirty-six cases of optic nerve disturbances, this difference was found eleven times. In the nine cases with canals below normal, the affected eye was always on the side of the smaller canal, while in the two cases with canals abnormally large, it was on the side of the larger canal, i.e., the eye affected was always on the side showing the greater divergence from the normal.

CONCLUSIONS.

The optic canal is normally circular and approximately 5.5 mm. in diameter. It may vary, however, from 3.5 to 6.5 mm. Excessive pneumatization about the canal causes distortion and narrowing, thus rendering it vulnerable. The size and shape of

the canal can be determined by *careful* radiography. The data, up to the present, indicate that there is great danger of permanent impairment of vision wherever a severe neuritis occurs in canals abnormally small, while with the same impairment of vision in a normal canal, spontaneous recovery may be expected. In the chronic conditions, the neuritis is thought to be toxic from some focus, such as teeth or tonsils. Neuritis, either acute or chronic, in the normal or abnormally large canals seems to be usually of extranasal origin.

This, if it proves true, should be of great importance in making a differential diagnosis between intracranial, multiple sclerosis, luetic and accessory sinus cases. While it is, of course, possible for any infection to reach the nerve through the system either by bacteremia or toxemia, the writer feels that canals of normal size are not especially vulnerable, and that either local treatment or the removal of some definite focus of infection is the more advisable method, rather than ventilation of the sinuses.

The conclusions of a preceding article still seems equally appropriate for this one: If future cases substantiate the findings in those already studied, it will mean that a canal of 4 mm. or less in a case of severe optic nerve involvement indicates the necessity for immediate ventilation of the posterior sinuses to prevent permanent atrophy, unless some other definite focus can be found. A 4.5 mm. canal gives greater leeway for study and investigation. Optic atrophy is less to be feared. A 5 mm. canal would probably recover from almost any *acute* attack either spontaneously or under local treatment. Then, if some focus of infection is found, diseased teeth or tonsils for instance, it should be removed to prevent recurrence.

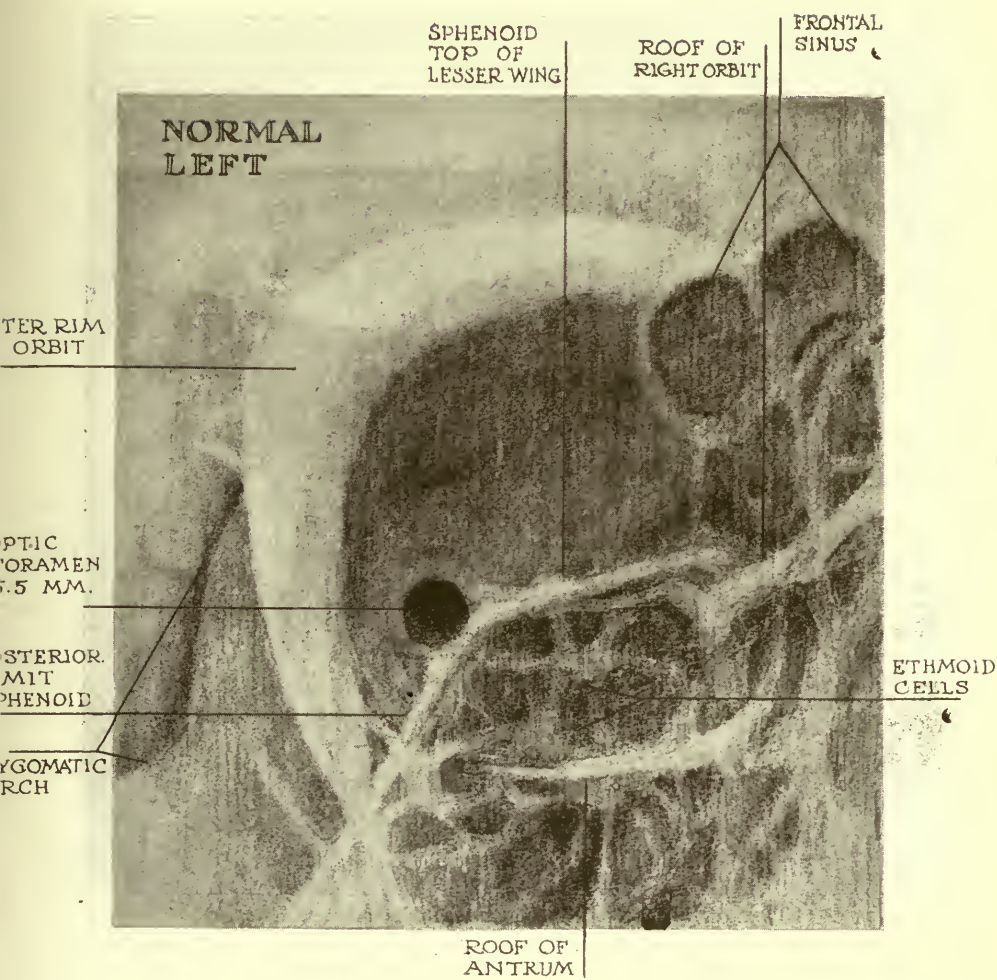


Fig. 1.

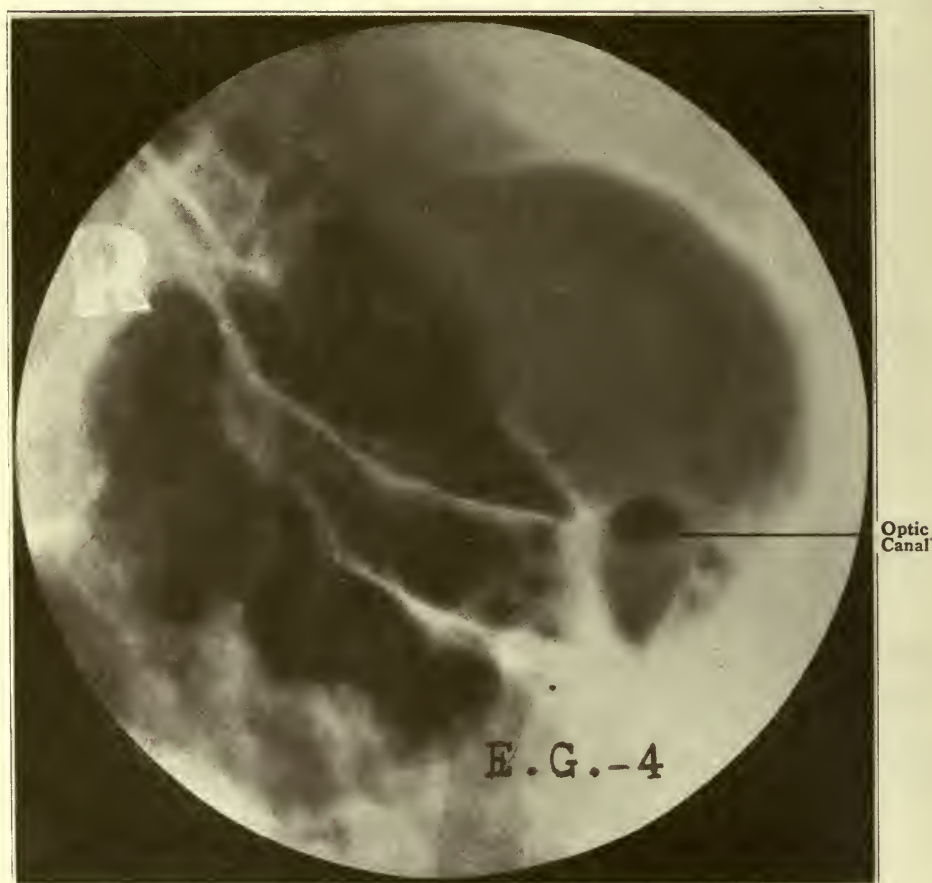


Fig. 2. Represents a small oval canal (4 x 5 mm.) in the correct position.
This case (No. 4) had marked optic atrophy.

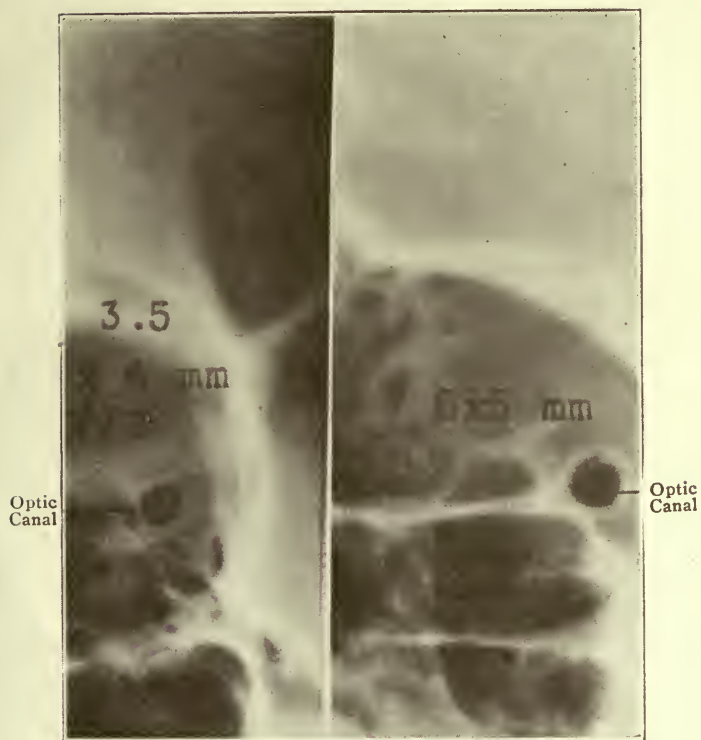


Fig. 3. Shows the comparison in size between the small and the normal sized canals. There was complete optic atrophy in this case (No. 27).

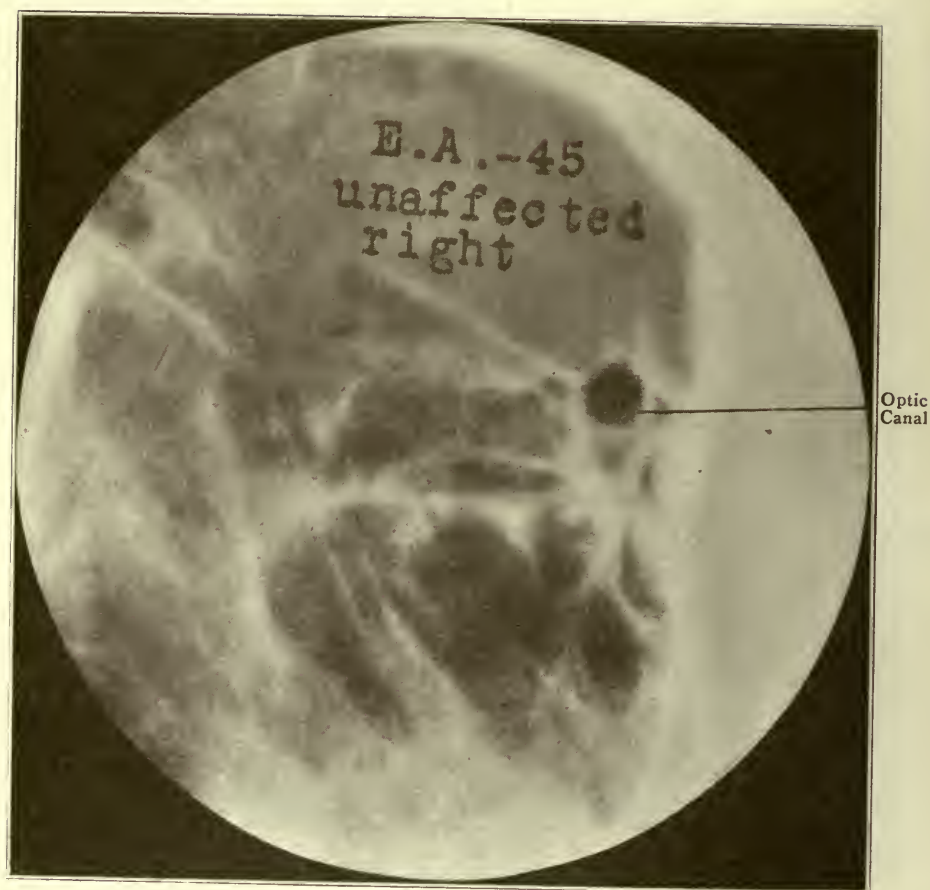


Fig. 4. This and the following plate represent the canals of Case 45.
This canal is of normal size.

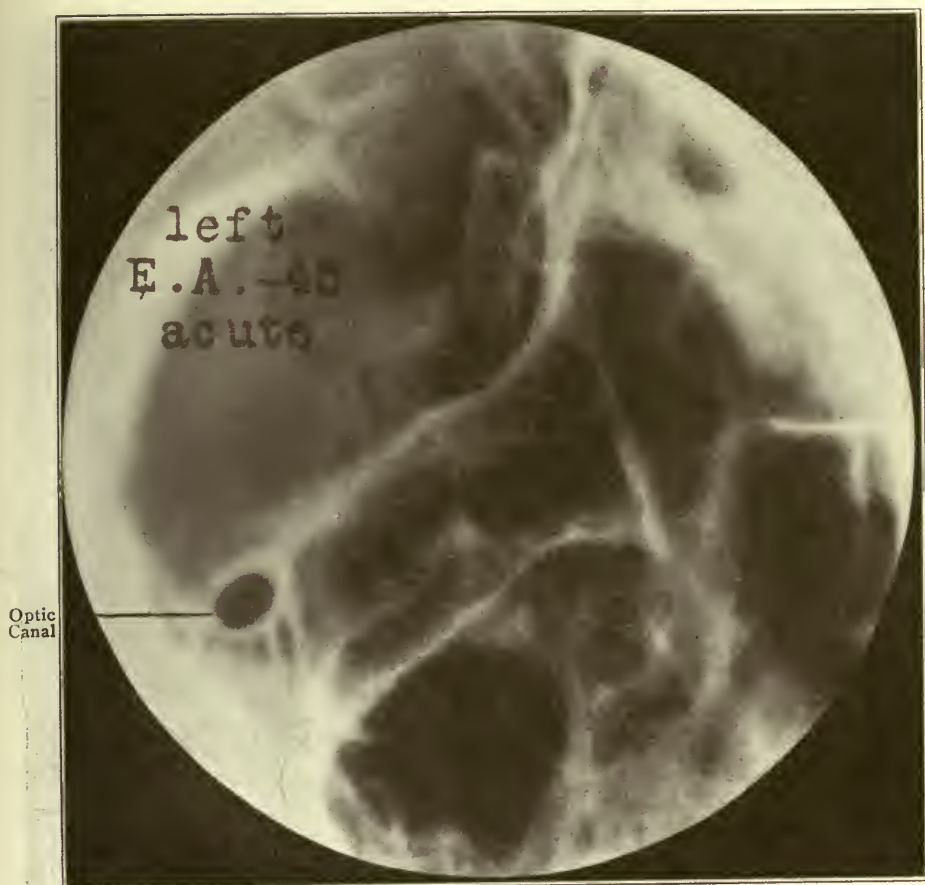


Fig. 5. Shows the affected canal of No. 45. It is oval in shape and 4 x 5 mm. Complete recovery followed removal of the tonsils.

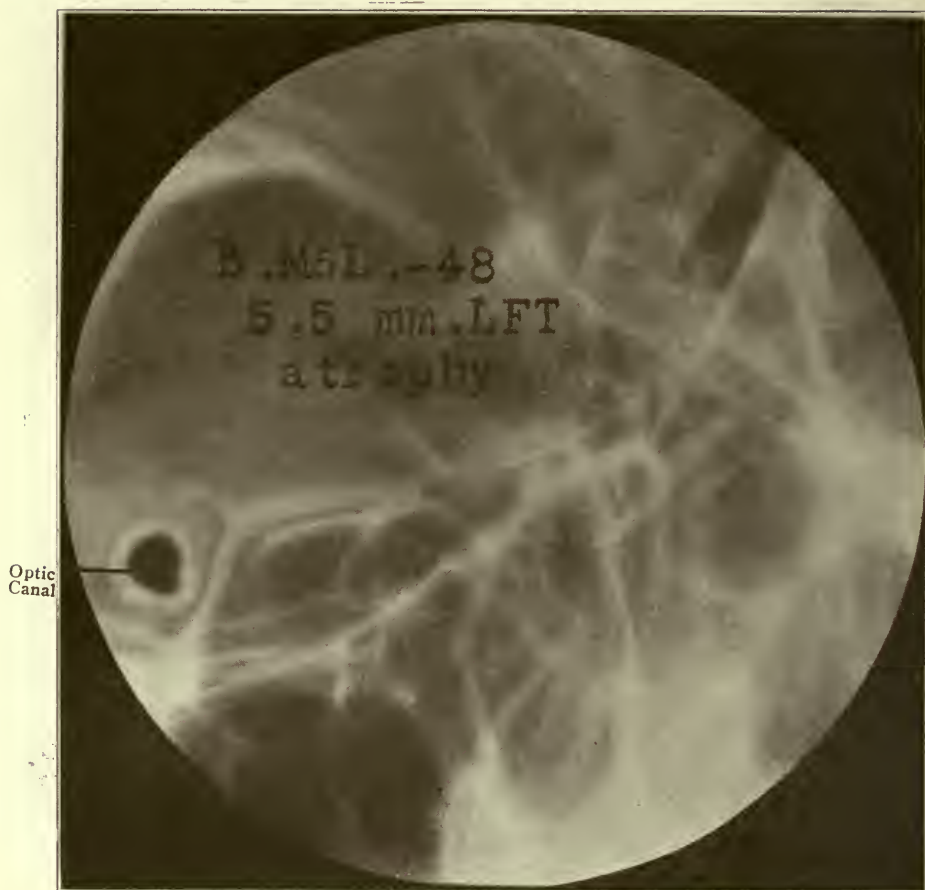


Fig. 6. Shows a somewhat distorted canal with what appears to be increased density of the surrounding bone. The atrophy in this case (No. 48) was probably from infected teeth.

DISCUSSION.

DR. H. W. LOEB, St. Louis, Mo.: The incidence of optic nerve trouble and sinus disease is not large, when one considers the great number of acute and chronic infections of the accessory sinuses. Naturally, the laryngologic and ophthalmologic divisions of the profession have been much stimulated by the reports of cases in which there was a possibility of recovery by operation upon the accessory sinuses. But as in other cases, it is necessary to study and discuss the etiologic relations and the anatomy, which by many is considered a lost art since physiology, pathology, bacteriology, biochemistry, and radiology have become more predominant.

The great merit of the paper and the observations of Dr. White is, that he has taken the anatomic findings in his investigations and has applied them to the subject at hand, and he has given us a most tangible method of determining when these particular findings are to be discovered. He has given a definite and very rational explanation of the occurrence of these findings and also the relation they bear to the pathologic conditions which we are called upon to face.

I feel that what Doctor White has presented cannot be discussed to any great extent, except by way of compliment and to express the decision on the part of those who are interested to follow his observations, to endeavor to substantiate, to amend, or perhaps do away altogether with the observations that he has made. At any rate, I think this is one of the greatest developments in the study of this particular relation, and one which should stimulate us to approach it not only from the anatomic standpoint but from these other standpoints.

DR. LEON E. WHITE, Boston, Mass. (closing): The following X-ray slides show the changes that took place in the optic canals and sella turcica in a case I saw last spring, referred to me by Dr. Cushing. The man had commenced to lose his eyesight a year ago, but Cushing could find nothing intracranial to account for his loss of vision. There were some changes in the nose through degeneration of the turbinates, and the sinuses were rather hazy. The man was anxious that something should be done to prevent total loss of vision. I took him into the hospital and had his tonsils and the middle turbinates removed and both sphenoids opened, but the vision continued to fail, and I would like you to see the changes that took place in the optic canals within six months. I think they are instructive. The man probably has brain tumor.

The lower outer quadrant of the right optic canal, which in the first slide is practically complete, shows an increasing area of enlargement and distortion, moderately marked in slide 2, and much more extensively noticeable in slide 3. The sella turcica in the first slide is within normal limit, i. e., 13 mm. in the horizontal diameter and 6 in the vertical diameter, while in the second slide, taken six months later, the horizontal diameter is 17 mm. and the vertical 11 mm.

THE LIFE HISTORY OF THE HUMAN LENS: ITS ORIGIN, ADOLESCENCE AND SENESCENCE.*

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INTRODUCTION.

In response to the kind invitation of your secretary to report to you regarding the human lens as revealed by the Gullstrand slit lamp, I must confess some hesitation, as I cannot give you much of my own observation, except in confirmation of the many new facts brought out by Alfred Vogt.

I have had the pleasure of expressing my gratitude to Gullstrand in person, in this same city, for giving us the most magnificent instrument of our ophthalmologic lifetime. Though it is the most scientific branch of the medical curriculum, ophthalmology has been at a practical standstill for 25 years. During this time one could not do more than master and verify the known fundamentals. Since observation has become possible with the combination of the slit lamp and binocular microscope, a new and heretofore unknown world has been disclosed. Alfred Vogt, now of Zürich, and Leonard Koepe are the pioneers in this new field of microscopy of the living eye.

The slit lamp as Gullstrand has given it to us is the best method for examining with focal illumination. It produces a sharply limited bundle of light, a minimum of halo and a well marked contrast. The eye does not suffer during prolonged examination, nor does its temperature become elevated. By narrowing the slit, we increase the power of localisation and determination of depth.

While the new facts regarding the human lens as described by Vogt are known to quite a few in this audience, they are not known as they should be to the great majority, hence the liberty of attempting to describe them.

You will then agree, it is hoped, that the chapters pertaining to the lens in our textbooks will have to be rewritten. Our present day knowledge of lens details dates back to the investigations of Becker, Foerster, Cohn, v. Hess, etc., some 4 or 5 decades ago. Considering the great importance of the subject, but

*Illustrated with lantern slides.

little advance has been recorded since their day. Almost immediately after removal, postmortem changes set in, and hardening fluids do not leave the lens intact. Now the lens can be studied in situ, at leisure, under magnification and intense illumination. Because of the fact that we may now see more than our fathers, it does not mean that there will not be new problems for the next generation to solve. Increased knowledge starts new problems. The method has its limitation. We cannot under the most favorable circumstances, with the pupil dilated ad maximum, see the lens equator, hence not the entire lens. In many instances one is limited to observation through a pupil dilated to 5 mm. or less. Of the two parts of the system, the illumination is the most important; an average magnification of 25 times gives us a better view than a larger one, especially in examination of the lens.

You will agree that it is out of place to defend here the slit lamp in our armamentarium. The same questions and objections are raised, which—if we may credit historical tradition—were expressed when Helmholtz had presented the ophthalmoscope. I consider that the slit lamp has at least an equal place, knowing that its value will increase with improved technic; that is, as much as the nitra lamp is an improvement over the Nernst lamp, so the microarc light has increased the light some 3.5 times over the nitra lamp, which allows more details than before.

Who will doubt that we are still in a condition as physicians where we want to know as much as possible about our patient and his sick organ? And when you are given an instrument, which enlarges your powers, do you want me to say anything more about it? You, who come here to these meetings, do not belong to the class whose members make it a point to pass through life with as little effort as possible, and to shine before the ignorant public with as small a knowledge as possible, and who can afford thousands for their automobile but not hundreds for an essential acquisition to their armamentarium. You absolve me from speaking about the technic, which certainly is a great deal easier than the driving of an autocar through a city.

The lens is a product of the ectoderm, influenced by the ocular vesicle. The optic cup and the lens originate independently. The first indication of lens development is an ectodermal thickening over the slightly invaginated ocular vesicle (15-18 days old

embryo). This thickening is slightly curved outward and a little larger than the ocular vesicle. This thickening forms the "lenticular plate", and is composed of one layer of high cylindrical cells, which are so crowded that the nuclei form in more than one layer. The mitoses are found near the free surface. A thin mesodermal layer lies between it and the ocular vesicle. While most cells end in straight lines at their base, some form a Lenhossik cone, which continues forming a fine fibrilla. These fibrillae form a contact between the lenticular plate, the intervening mesoderm and the wall of the ocular vesicle. Some 5 days later, the lenticular cells have moved inward, and the originally slightly convex form has made place for the concave "lenticular groove". This is thickest in the middle, which makes the "groove" flat rather than an excavation. It contains 3-4 cell layers. Some few loose cells are seen on the "groove". The basal cones have become more numerous, with a tendency to bifurcate and form two fibrillae. As the invagination of the ectoderm increases, the lenticular groove becomes the "lenticular sac", which is ventrally deeper than dorsally. In this newly formed cavity loose cells are also found. They originate from the wall of the lenticular sac, which also shows cellular mitoses near its neck. Many of these cells show signs of degeneration and must be considered as evolutionary detritus.

The lenticular sac then develops a tendency to constriction. Its distal end is still in free communication with the amniotic cavity through the "lenticular pore", a little more ventrally than the center of the distal wall. Ectoderm and distal lens wall together form folds, which fuse at the margin of the opening, the dorsal fold being longer than the ventral. The constriction becomes total and the "lenticular vesicle" is pinched off of the ectoderm. Numerous homogeneous, strongly reflecting bodies are here seen. They are probably a degenerative phenomenon. In the beginning, the vesicle is more triangular than round. With continuous growth the number of cells diminishes. When the constriction is complete, mesenchyme develops between the vesicle and the ectoderm. The Lenhossik cones are still present. A distinct differentiation between the distal wall, which develops into the lenticular epithelium, and the proximal wall, from which the lenticular fibers originate, is now apparent. The cells of this proximal wall become elongated and form a cushion ("Polster"), which bulges into the cavity. The middle fibers are straight in direction from behind forward, while the peripheral ones form a curve with the concavity outward. The nuclei of the fibers are

not in one plane. After the fibers have attained a certain length, division figures are absent. The lenticular vesicle has now become rounded. The growth of the fibers has reduced the cavity to a crescent, and there are no loose cells found in it. The epithelium of the spherical lens is thinnest in the center of the anterior surface, and increases in thickness towards the equator. The nuclei of the lens fibers lie more toward the anterior wall, because the fibers have grown more backward than forward. The growth of the lens from now on takes place by apposition. The epithelium stops at the equator, where the cells become arranged into meridional rows. At the posterior end of these rows the cells develop into fibers, "the bow of nuclei", which bending in a sharp curve posteriorly turn inward and forward. The growth of the lens proceeds from the epithelial border, new epithelial cells continuously developing into lens fibers. The nuclei of these cells move toward the interior of the lens, so that one finds a zone along the equator where there are many nuclei within the lenticular substance.

The lens sutures begin to form at the end of the third month, and at the fifth month the lens has developed a star with 3 radii. The posterior suture forms first, beginning as a short linear slit. Later this bifurcates. The sutures develop because of the fact that the central lens fibers are shorter than the outer ones; therefore the posterior ends of the outer fibers overlap the shorter central ones. Where the posterior ends of the longer fibers, which come from above and beneath, meet, a horizontal cleft arises. This cleft increases in depth as the peripheral fibers with their posterior endings overlap more and more those within them. The cleft continues to the posterior end of the lens, so that the posterior end of the cleft is formed by the longest, most posterior fibers. The anterior vertical cleft originates with the posterior horizontal cleft. These two clefts are perpendicular to one another. The fibers which reach the posterior lenticular pole, end anteriorly quite a distance from the anterior pole, above and below. On the other hand, the fibers which lie near the anterior pole direct their posterior endings towards the sides of the pole. Fibers of the same age are similar in length. We therefore, at the time of birth have a double star, formed by 3 slits, which unite in the center, the anterior in the shape of a vertical, the posterior in an inverted Y. The lens grows continuously during life. The lens stars become more complicated; the secondary branches do not reach the center. The original vertical and inverted Y can always be seen, and as they mark the outer parts

of the embryonal nucleus, we are able to differentiate this part of clear lenses as the "embryonal nucleus". The Λ on the posterior surface, with its concavity forward, nearly always reflects the light stronger than the anterior vertical Y. A continuation through the centers of those two stars definitely localizes the posterior lens pole.

The lens is not composed of a homogeneous substance. Lenses of even young children show zonules of different refraction. In adults, one concentric zone becomes quite prominent, and it reflects the light more strongly. This zone at the end of puberty is just beneath the capsule. The lens continually growing by apposition of newly formed fibers, the older layers become more centrally situated, and this part of the lens forms what may be called the "adult nucleus". The material within gives off water and becomes sclerosed, and this contraction is seen as a peculiar beautiful relief-image, originating from the lens sutures as a sort of "haute plateau". The visibility of this new nucleus proves that sclerosis has taken place within it. Its development begins as an axially situated vertical suture ridge. Later, ridges, banks, and knobs may be seen. During ordinary examination, in some cases, the impression of a well marked relief may at times be noted. Transillumination with the loupe mirror, if showing spots at that region, may prove the presence of vacuoles on the outer part of the nucleus. The relief-image at the posterior side of the lens presents itself as the negative of the anterior relief-image: prominences appear as depressions, etc. The relief-image is best seen when the axis of the microscope is perpendicular to the direction of illumination. Dilatation of the pupil is unnecessary. It is invisible by transillumination, and is most pronounced in the anterior center, but does not affect visual acuity.

Sclerosis of the lens is, however, not limited to this adult nucleus. Though the cortex may be sclerosed, the relief-image may be seen. A distinct difference in refrangibility must cause this visual phenomenon. However rugged the appearance of the relief may be, it does not interfere with the central visual acuity, and has no relation to cataract formation. Sclerosis of the lens may be a process of conservation. It protects from disintegration. Opacification of sclerosed areas is never as intense as that of the cortex. Lens sclerosis may ultimately reach the capsule, and in such a condition we speak clinically of a large nucleus. A nucleus of this kind is entirely different from what we have found with the slit lamp as the "adult nucleus". The sclerosis of this nucleus will, to a certain extent, counterbalance the flattening

of the lens and preserve the average refraction; it is ultimately the cause of presbyopia. Vogt found that the strongly reflecting surface of the adult nucleus recedes from the lens surface in the course of years in a way similar to contusion cataract. The axial thickness of the cortex increases relatively to the entire thickness of the lens, though an absolute increase of the cortical thickness was also found with a simultaneous decrease of the nucleus.

With the aging of the lens, its inner, diffuse reflection and dispersion increase through the increase of the density of the lens substance, and opacity and yellow coloration develop.

Each structure in the body has its proper vitality, and nothing prevents us from assuming that each cell has its definite duration of life; or otherwise expressed, certain cells may degenerate at a certain period of life, with entirely normal conditions of the surroundings and without recognizable external causes. The primary cause must be looked for in the germ plasm, and is therefore hereditary. We need not presume that an organ ages in its entirety at the same time. Senescence may therefore appear in different aspects.

Taking the body as a whole, we see for example, baldness or decay of teeth or wrinkling of the skin in different aged individuals, without being obliged to assume causes other than that some organs age earlier in some people than in others, and we "explain" this by saying that it is due to heredity. So in the lens, some parts change earlier than others, and we often find this verified in different members of the same family.

Thus may be seen the earliest form in which senile lens degeneration appears. It starts soon after, even sometimes during puberty, in concentric thin layers anterior and posterior to the equator of the adult nucleus, where the second and third part of the lens radius meet. Vogt found it in some 20% of healthy people, and called it "cataracta coronaria". This was called formerly cataracta coerulea, or viridis, or punctata, and considered quite rare. Its characteristics are the development of club or raisin seed shaped opacities. Towards the center, circular discs or annular spots may be found. The bases of the clubs lie toward the equator. They often coalesce. If seen from above they form lines. If they are thin, they are seen in different colors according to their depth, the blue ones being the deepest; white if they are thick; green in the intensely yellow lens; and if seen with reflected light they are brown. It conforms entirely in location and form with the type Becker and Förster studied as cataracta senilis incipiens. As it is not found in children under 15 years of age,

though very common after that age, this form of cataract is an important precursor of senile cataract. It is often present in cataract, which also shows spokes in people of over 60 years of age. With the loupe mirror, these opacities are often mistaken for vacuoles. Later in life it may be combined with water fissures. Its evolution is slow. As I have stated, it is often found in many individuals of the same family.

Another common form of senile cataract begins as multiple points of irregular, angular shape and size, dispersed through the entire cortex, but the densest deep in the cortex towards the equator, where they form a peripheral, concentric layer—opacifications. They do not interfere with the vision so long as the pupillary space is not much affected. Its first appearance is seen as broadening of the adult nuclear zone, which also shows an increased reflection.

Still another form of beginning senile cataract is seen in the formation of “water fissures”; these originate through imbibition of fluid, which separates the lens sutures and fibers. First these slits are clear; later they become filled with swollen lens material and globules (the Morgagnian globules or myelin drops); after this, opacification may be seen in the routine examination as spokes. Some spokes arise directly between fibers. The water fissures are dark with incident light, with whitish borders on account of increased diffuse reflection, probably through changes in the adjacent lens fibers (imbibition). They have the most irregular forms, and are not limited to distinct zones, although mostly found in the middle and deep layers of the cortex, sometimes directly in front of the adult nucleus. Their rate of growth is not definite; they are observed to remain for a long time unchanged or they may grow rapidly. The imbibition with water depends on inner causes—changes within the lens substance. It is often a symptom of cataracta intumescens. As soon as the inner cause passes off through destruction of the dead fibers, which causes the swelling, the water fissures retrograde and the mature cataract is present with a smaller volume.

The onion like structure of the lens manifests itself in the following manner. “Cataracta cuneiformis” is a layer like opacity, sometimes found in more than one layer, distinctly seen at some distance from the anterior lens capsule, in the middle cortical layers, with its base toward the periphery, extending centrally in roundish lines or sharper wedges, which latter may continue in spokes. It is found in the anterior and posterior cortex, where the opacities meet at the equator, or end toward the equator in

a regular concentric line. It is found mostly in advanced age. The color is white, or yellowish in the posterior cortex, due to the coloration of the lens substance anterior to it.

Connected with this cuneiform cataract, but also occurring independently, may be seen fine parallel lines—the “Lamellar splitting or separation”—in the same region, and axially from it, usually in the inferior nasal quadrant. These opacities are arranged from below, temporally toward above, nasally. Sometimes they show another direction. The separation can bend in the direction of the fibers. This separation is due to water imbibition. When the “system” is crossed by a seam, the lamellar lines seem to sink into and be attached to it, or the lines bend over the seam in their forward progression. The anterior nuclear surface, especially in advanced cataract, can also show this lamellar splitting, which may produce a spiderweb arrangement.

The onion like architecture of the lens may also be shown by linear concentric opaque zones, the expression of zonular opacification, yellowish in color, around the equatorial nuclear region, deeply within the cortex.

The slit lamp has done much to give us a better understanding of “posterior saucer like cataract.” Subcapsularly, the latter shows a layer of crystalline densification with vacuolisation. As it progresses, it increasingly gives an impression of pumice stone. It is often more developed peripherally than axially, and is usually combined in the equatorial, deep cortical region with old opacities. The nucleus is also often affected. It is that form of cataract where the visual acuity does not correspond to the amount of light reflected from the fundus. Slowly the density increases and passes over into the anterior cortex, leaving the greatest part of the axial cortex and nucleus relatively transparent, so that the pupil looks black. This form of cataract must be differentiated from the secondary or choroidal cataract. Here the progression of the density is forward, and the coloring of its chagreen is characteristic. This form of cataract is slow in ripening, though it is not necessary to delay operation. Its prognosis is good. In most cases the cortex is also sclerosed, so that it is delivered with the nucleus. Such cataracts were often found small in size by Vogt.

Observation has also shown the frequency of nuclear cataract. As the physicochemical constitution of the nucleus differs essentially from that of the cortex, so the cataract formation of the nucleus begins in another way. It shows an increase of the in-

ternal diffuse reflection of the light. This opacification begins around the dark interval in the embryonic nucleus. After this nucleus is entirely involved, it may still be separated from the periphery of the adult nucleus by a clearer interval. This may later become involved. A diffuse opacity may then be seen, in which no separate elements are distinguished. This condition seems to be peculiar to the nucleus of adults and old people. The embryonal sutures are seen within as white lines. Even if the visual acuity is diminished to 6/60, the nuclear cataract can be seen, which is explained by the form of opacities, which have a cloud like appearance, in contradistinction to the rather large globular destruction seen in the cortex.

The normal lens shows its formation in an interesting way. It is composed of 3 parts: the embryonal nucleus, which is the lens as it is at birth; this becomes surrounded by the adult nucleus, the lens as it is at puberty; and this again enveloped by the ever increasing cortex. Each part has its own individuality, as is shown by its characteristic forms of cataract, which has a practical and useful side in so far, that we can decide if some opacity is recent or old, congenital or acquired, stationary or progressive.

The embryonic nucleus shows a very common type of cataract—the “anterior axial embryonic cataract.” It is found in different degrees of extension around the anterior embryonal star. It is seen to be composed of separate or coalescent, strongly white reflecting points surrounded by a halo. They probably are connected with the loose cells described in the ontogeny, and would therefore point to some disturbance about the second month of embryonic life. This circumscribed cataract is very common (according to Vogt in 25%) and can be seen with the pupil undilated.

Within the embryonic nucleus we may also find what is known as “*cataracta stellata*”. This form of cataract presents linear or streaky opacification, often of bluish or blue-greenish color, rarely white, along the anterior and posterior sutures. It is often combined with small, round or oval discs of the same color in the nuclear periphery. It can also be found combined with “*cataracta dilacerata*”, which latter shows a moss or grill like branched opacity, extending in concentric layers. We may also find “*cataracta pulverulenta*”, composed of diffuse or concentric points and dust like opacities. This form of cataract is seen in different degrees of lamellar involvement. All these forms of cataract of the embryonic nucleus seem to be stationary.

The adult nucleus may also show "zonular cataract," also known as perinuclear or lamellar cataract. One of its commonest forms shows an external zone with radial spokes, separated by a clear interval from a deeper layer, which latter is formed by points. The spokes pass peripherally directly, or after interruption, and form the riders, which usually lie within the same lamellar zone. Clear radial slits at the equator, filled with normal lens substance, are not rare. The embryonic nucleus often participates in this form of lens clouding. Isolated riders are often found independently around the equator of the adult nucleus.

It is of course entirely out of the question to describe the life history of the human lens exhaustively in the time allotted; many details anatomically and other have not been touched upon, others only very cursorily. I did not speak of the anterior and posterior lens chagreen, which in advancing age shows a less regular graining, making one think of the depressions and elevations of the orange rind, or of the increase of the chagreen globules in old age, or of the color reflex of the anterior capsule, of the subcapsular vacuolar layer anterior and posterior, between the epithelium and adjacent fibers, or of fine punctuate coloring of the anterior axial sutures. Nor did I mention the visibility of the lens epithelium, or the signs of continuous growth, which are seen in the recession of opacities after concussion, or the many forms of zonular cataract, or all these forms we include under the term: "cataracta complicata", or the increase of color of the lens, or the chemical changes which go on while life progresses. What I hope to have conveyed is, that when speaking of cataract, we must henceforth definitely specify its form of opacification. We will have to consider in the different forms the time of their genesis, and the tendency to progression or its definite absence. We must decide as to the effect of possible medical interference, and classify cataracts as to their heredity, frequency and time of appearance. When this has been accomplished, ophthalmology will be free of many phantoms"—those of "predisposition to cataract", the hypothesis of exogenous noxious substances, the theory of "chemical nutritive disturbances", and "ultraviolet rays"—the latter according to Vogt, the "Prügeljunge (Scapegoat) of modern Ophthalmology".

A STUDY OF THE CORNEA WITH SLIT LAMP AND MICROSCOPE.

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The combined slit lamp and corneal microscope has amply justified its existence. The workers with it are increasing in number and daily observations prove its value. No method compares with it in ease of application or range of usefulness.

The source of light, the position of the observer and the part examined must all be controlled to secure a clear corneal picture. The light is best thrown from the temporal side of the eye, and must be so focussed that an image of depth is obtained. Those who have worked with the lamp appreciate what this means, but to others who are beginning, or who have had difficulties, this is most important.

The way we find this slit of light most easily is to focus on the margin of the iris; then, without moving cornea or lamp, the observing tube is turned a few degrees to the temporal side, when the gray band is immediately outlined. If the true parallelogram is not distinct, it can be made clear by adjusting with the thumb screw at the end of the lamp holder. By focussing to the side of the brilliant gray zone, the object is transilluminated. All corneal diseases must be studied by direct and indirect light. The low power is the one of choice for the gross examination. The highest magnification is more difficult to use and should be reserved for minute detail.

With a sharp focus, we appreciate the glistening anterior surface and the less clearly defined posterior surface; between these there is a zone representing the thickness of the tissue. If the angle between the illuminated region and the observer is great, we have a mirror like reflex which is epithelial, but if more acute, we have a diffuse gray reflex. When the correctly focussed light strikes the cornea at an angle of 40° and the observer focusses on the lighted field, he will see the true slit. It is, however, necessary to continue focussing the light and observing tube as they move across the cornea, which maintains a fixed position, otherwise there will be a marked broadening of the band of light with less detail.

We will consider the five layers of the cornea: epithelium,

Bowman's membrane, stroma with nerves, corneal corpuscles and lymph spaces, Descemet's membrane and the endothelium. The limbus presents changes which differentiate it from the remaining portion of the cornea.

Study of the epithelium reveals two normal pictures, tears with the included dust, easily proven to be superficial by their disappearance or motion on winking, and dew like changes; the latter, best seen at the limbus, consists of minute droplets. The line of demarcation between physiologic dew and pathologic stippling is at times most difficult to draw. There are all degrees of edema of the epithelium from minute stippling to large blebs and bullæ, as in herpes and dendritic ulcers. These epithelial elevations are irregular in outline and are best seen by transillumination. Stippling has been found after the instillation of drugs, such as cocaine, in the various stages of glaucoma, keratitis disciformis, in beginning interstitial keratitis, after exposure of the cornea, such as follows Gasserian ganglion excision, and in many other conditions. If bullæ rupture, the water soaked epithelial layer is gray and elevated, with sharply outlined margin. The branched form of the dendritic lesion is recognized from its incipency to its cure. Epithelial cuts, lacerations and abrasions are so clearly defined, that it is unnecessary to use fluorescein or any other tissue stain. The common epithelial abrasion which follows the scratch of a fingernail shows a loss of tissue with an irregular elevated outline.

Some very interesting observations have been made in herpes ophthalmicus. In one type, the entire corneal surface was stippled, with infiltration of the stroma and a collection of cells on the endothelium. As the infiltration lessened in extent, the stippling gradually disappeared, and finally only superficial stromal opacity remained. In another form following trauma, there were three distinct changes. In the superficial stroma, there were several more or less rounded infiltrations; in the deep stroma, many larger infiltrations; and, associated with these, many endothelial deposits, irregularly outlined, thick, brownish in color with distinct brown granules.

After the study of the superficial changes, it is best to consider vascularization. There are two types of vessels, the straight and the tortuous. The straight usually branch dichotomously and are most frequently found in the true stroma. The tortuous, which vary in size and degree of tortuosity, may be in any portion of the cornea. The most practical subdivision is the location of the vessels, especially considering the source of their blood

supply; the superficial, the deep and those from the iris. Clinically, the combination of the three is not rare, and the association of the superficial and deep common. The extension of blood vessels to a phlyctenule illustrates the superficial, the salmon patch of interstitial keratitis the deep, and a vascularized, adherent leucoma the iridic.

The pigmentation of the cornea is of sufficient value to warrant special notation. Hemorrhagic extravasation may be anterior, posterior or confined to the stroma. Purulent infiltrations may be general or local. Siderosis may present as a fine brown or even a dense, grayish-brown sheet. Rust particles may be found in all layers of the cornea, if the iron body on its passage into the globe deposited pigment in the track of entrance. When the foreign particle is retained within the eyeball, fine pigmentation may completely infiltrate the endothelial layer. For many years it has been noted that old corneal scars tend to show a gross yellow color. This coloring may be in part of the opacity, or may seem to be throughout the scar. This is not to be confused with the brown, deep line characteristic of some cases of conical cornea. Endothelial pigment deposits may be seen in melanotic sarcoma of the uveal tract.

Bowman's membrane cannot be differentiated by the slit lamp. Pathologically, however, two lesions can be diagnosed—rents and folds. In the former, it is possible to focus on the margin of the opening; in the later the thickening can be seen sometimes as radiating lines.

Although the untrained worker finds the study of the stroma unsatisfactory, investigation of this part has been stimulated and new facts accumulated. From the slightest superficial infiltration to complete destruction, all degrees of change may be discerned. It is instructive to record the clinical course of a severe ulcer, where in the active stage the entire thickness of cornea in the region infected is greatly swollen, and the infiltrating cells demonstrable. This cellular infiltration is followed by tissue loss, reparative process, and resulting opacity with blood vessel production and occasionally with crystal formation.

In the earliest stage, there is an increased grayness of the stroma, through which the light does not penetrate. As the process increases, infiltration usually extends in depth and over a greater surface, accompanied by vessels if the epithelium is intact. When the epithelium is lost, stromal infiltration may be unaccompanied by new vessels. It is possible to see the clearing of dense infiltrations as well as the organization of others. If cal-

cification is marked, the epithelial layer is frequently lost, evidenced clinically by a congestion and irritation of the eye. This exfoliation can be distinctly outlined, as well as the depth and intensity of the opacity. Irregular scars resulting from interstitial diseases may be marginal, extend as a band across the cornea, corresponding in shape and position to the palpebral fissure, or they may be so dense that no differentiation of layer is possible. This matting of layers is seen particularly after complete destruction, such as follows an ulcer from pneumococcus or Neisser infection.

One of the most important landmarks for the accurate determination of location in the corneal substance is the position of the nerves. They are in the middle or anterior third of the stroma, and extend from the periphery, usually carrying the sheath a short distance. They are seen as gray white threads, straight or wavy. The ganglia may be outlined at the intersections. In inflammatory processes, either the nerves are greatly increased in number or are more visible, for it is certain that under such conditions they seem to be more numerous.

Interstitial keratitis, a disease of perplexing individuality, shows a variety of forms from its beginning to its end. Some cases show irregularly round areas of infiltration in the superficial layers, without vessels extending beyond the limbus. We have seen this avascular stage last eight weeks, to be followed by intense generalized vascularization. Others start with faint stippling, while still others begin with a slight infiltration surrounded by vessels, and progress by the extension of the vascularized opacity. The superficial and deep stroma may be involved at the same time. In most cases, there are endothelial deposits, usually dense brown, but occasionally, even in well advanced cases, only slightly pigmented. At times, adherent to the endothelium is a complete network of fine, gray fibers, which form irregular meshes, containing within their structure innumerable, brown masses. There is no doubt that the blood vessels functionate even after all inflammatory symptoms have disappeared. By transillumination, the blood circulation is always demonstrable.

Diseases of Descemet's membrane have been classified in various ways, described under numerous titles and treated most haphazardly. Changes in the membrane are wrinkles or ruptures. The wrinkles may be short and barely perceptible, or folds of considerable thickness may extend completely across the cornea. The whole membrane may be irregularly folded upon itself, show-

ing gray lines, the dominant direction of which may be vertical, horizontal or crisscross. These folds may be limited to the zone of infiltration; they may result from healing scars, from trauma, or follow changes in intraocular tension. The differentiation between a rupture and a fold is made by the light reflex.

The endothelial layer is usually observed as an oval, brownish-yellow in color. Individual cells are demonstrable, and deposits on them easily outlined. There are certain physiologic changes, particularly the Hassel and Henle warts, which are to be considered before pathologic changes may be understood.

Endothelial deposits are studied by noting the gross shape of the area covered, the size of the individual deposit, its color and its life history, the latter including duration, end result and response to treatment. Although the shape of the area covered by these deposits has for many years been considered triangular, with the larger deposits below, we believe that clinically this is not the most common distribution.

There may be a single deposit. This may be thin and clearly visible with rounded outline, or be star shaped. It may be so small as to be a microscopic speck, or so large as to be grossly visible. The color ranges from white to almost black, showing as gray, slightly stained yellow, or brown. The deposit may be uniformly of one color, such as white or dark brown, but often it shows the granules of the brownish pigment on the lighter base. Although at times only one deposit is found, often there are several, and not infrequently the entire membrane is studded with them. Even when deposits are over the whole surface, they may remain separate. When they become confluent, they appear as elevated, wavy ridges. The deposits have been observed to disappear gradually, but some have remained unchanged for months. The associated conditions are iritis, both the acute and chronic, keratitis, choroiditis, glaucoma and trauma. The end result seems to be materially influenced by treatment, particularly the tuberculous under tuberculin, the luetic under mercury and iodids, focal infections following the removal of the infected tooth or tonsil, and the appropriate care after trauma. The deposits observed in sympathetic ophthalmia have, as one would expect, been little changed, even by vigorous treatment. Pigment in glaucoma does not seem to be absorbed, and is always increased after operation, this increase being not only in the number but also in the size of the deposits. Observations seem to prove that round or star shaped white masses are most suggestive of tuberculosis, dark granules of intraocular pressure, white deposits with faint

pigment of focal infection, and the intensely pigmented ones of chronic involvement.

In a cut through all layers of the cornea, if apposition be inexact, it is possible to see the loss of epithelial cells, the curling of Bowman's membrane, the distorted lamellae of the stroma and the furled Descemet's membrane with loss of endothelial cells. If iris is incarcerated in this wound, there will be some uveal pigment adherent to the scar, even if later the iris is replaced. In a dense adherent leucoma, iris tissue is always visible, the scar frequently elevated and sometimes very thin, particularly after traumatic prolapse of iris or after cataract extraction.

The limbus, with its conjunctival overgrowth, presents many characteristic features. It is demonstrable in health that many blood vessel extensions seem to be closed, but on the least irritation, such as the rubbing of the eye, these vessels become patent, and the blood is seen to circulate through them. The degree of vascularization is extremely variable, and the differentiation between normal and pathologic is sometimes made only after the continued observation of the given eye.

Vessels may extend as single loops, there may be a band of them, or they may extend in several layers. In subconjunctival hemorrhages, these vessels are engorged and extend farther into the cornea. The development of a pterygium, starting with a single blood vessel with translucent, yellowish-gray advancing margin of conjunctiva, is more thoroughly understood and better appreciated than ever before. We have observed this overgrowth of gray opacity with projecting blood vessels in a case of chronic uveitis, where an irregular arc extended more than 3 mm. into the cornea. The marginal ulcers, with increased vessel formation, may heal leaving a thin, irregular, grayish opacity, to which a single vessel may continue supplying blood. There may be innumerable, small, superficial, more or less rounded infiltrations, which have a tendency to recur. With the extension of pannus, not only are the conjunctival vessels traced in their numerous loops, but the individual loop is found to center around a superficial ulcer. This ulcer has a more or less crescentic appearance, with elevated gray border.

Pigment between the limbal vessels is not unusual in the elderly. It is also found after prolonged nitrat of silver treatment. In many corneal scars there are black pigment deposits, and in others minute white specks. The cornea in the region of a trephine opening shows elevation, infiltration and edema, with

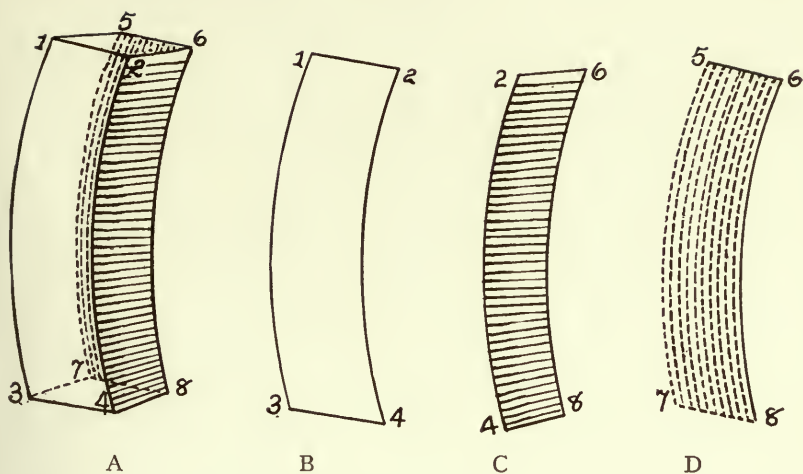
pigment granules not only in the corneal scar, but also in the conjunctival cicatrix.

In the tuberculous infiltrations extending from the margin, we observe superficial stippling, and separated from the stippled zone, a deep infiltration. This deeper area has an uneven, more or less rounded margin, which may consist of heaped up cells, or may have a thin, fringe like edge. Within this opacity may be denser, usually rounded patches of infiltration. Both superficial and deep blood vessels are demonstrable.

The marginal phlyctenule shows an elevated, edematous, indistinct outline, with a translucent boundary and innumerable vessel tufts, some of which may look like hemorrhagic dots.

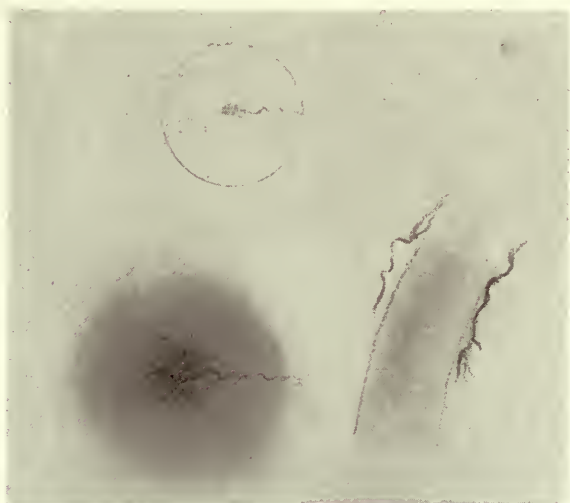
An arcus senilis is always separated from the limbus by a zone of clear cornea, so, remembering this, there is no possibility of confusing a corneal scar with it.

In conclusion, there is no method of corneal study which gives as much detail and as valuable information as examination by the slit lamp. Lacerations of the epithelium, abrasions and small foreign bodies are accurately located and the extent of involvement determined. The clinical course of any corneal disease is better understood, and complications and sequelae anticipated. The depth and extent of corneal opacity are exactly fixed, and proper operative procedure considered. The many diseases that may cause stippling are differentiated. Interstitial keratitis manifests itself in an infinite variety of changes, not only in mode of onset and clinical course, but also in the final clearing of the opacities; and, finally, endothelial deposits afford information regarding the cause of the uveitis, and materially assist in the direction of therapeutic measures.

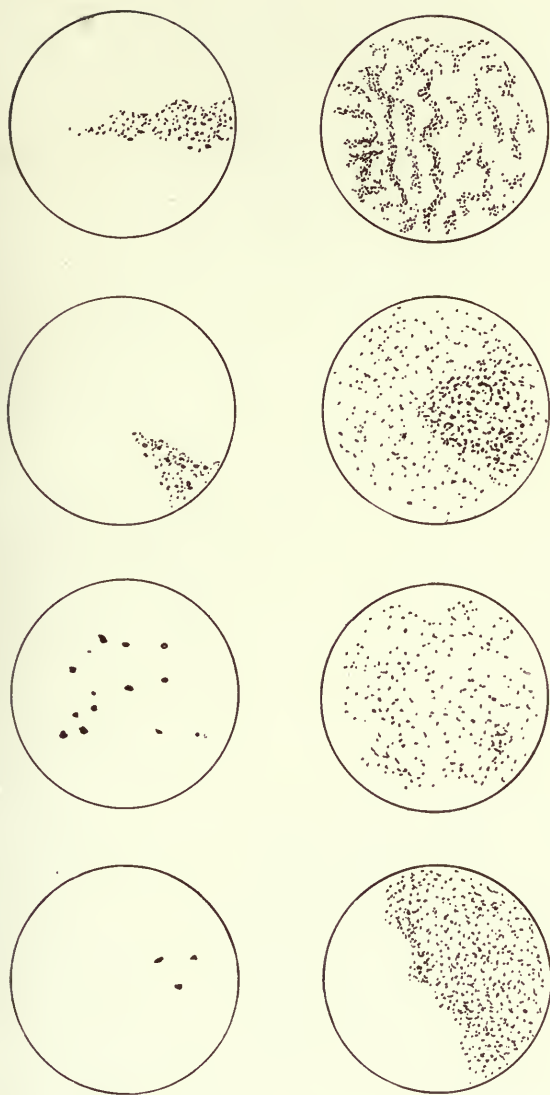


"A" represents a true parallelogram, "B" the anterior surface, "C" the depth of the tissue, "D" the posterior surface.

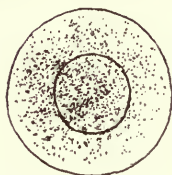
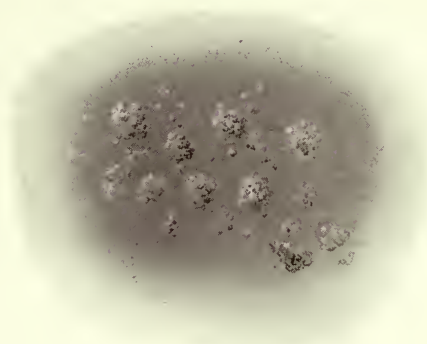
By combining B, C and D, and carefully noting the position of each, it is possible to definitely determine the site of the lesion. In the cornea "B" would represent the anterior surface, "D" the posterior surface, and "C" the cross section between.



A drawing of small blood vessels which were epithelial and endothelial.



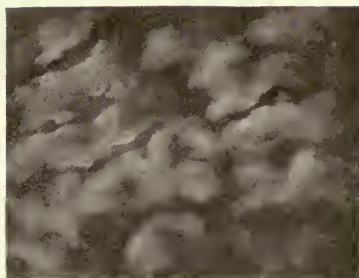
Illustrations of some endothelial deposits with relative size and distribution. Particular attention is drawn to the infrequent triangular arrangement, to the fact that the triangle may only represent an increased number of deposits, and that the deposits may be arranged in heavy ridges.



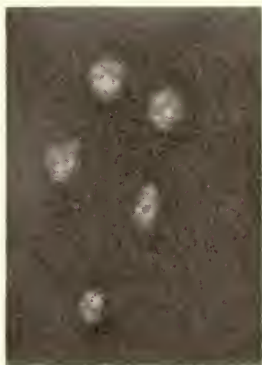
These granular white deposits were found in a woman, 45 years old. The only etiologic factor was an apical abscess. The removal of the diseased tooth was followed by recovery and complete disappearance of all deposits.



A male painter, 51 years of age, who has always been deaf, had a sore eye for a month. The deposits were irregular in outline and thickness, with many faint brown granules. No etiologic factor found except possible lead poisoning.



Ridges of white exudate on the endothelium, with wrinkles in Descemet's membrane, in a woman of twenty-five. Vision was reduced to motion of the hand at three inches. She had a positive tuberculin reaction and infected tonsils.



Endothelial deposits with irregular margin showing much granular, brownish pigment, in a case of herpes ophthalmicus.



Two endothelial deposits in a man, 45 years of age, with negative findings except two apical abscesses. Recovery has been complete. The principal deposit shows a nebulous haze surrounding a thick white elevation, with faint brown granules of pigment. The smaller deposit is as yet free from pigment. This may be the result of the age of the deposit, or because it is so thick that the fine granules which may be contained within it are not evident.



Typical tuberculous endothelial deposits, thick, white, irregular in size and outline, with no pigmentation. The patient was a boy, 18 years of age. The eye cleared entirely under hygienic surroundings supplemented by tuberculin injections.

DISCUSSION OF DR. BLAAUW'S AND DR. BEDELL'S PAPERS.

DR. HARRY S. GRADLE, Chicago, Ill.: Owing to the carefulness of their observations and the thoroughness with which they are recorded, the authors place us in the position of having very little to say.

In the past, before modern methods of examining the cornea with the slit lamp were introduced, we were very confused as to the classification of corneal diseases. We thought we knew too much about the cornea, because every disease of the cornea was visible to the eye, aided by the means at our disposal, and in consequence every stage of the disease could be studied in its entirety; but since the slit lamp has been added, and such careful observers as Bedell have spoken and written of the various corneal conditions, we are more confused than ever as to their classification. Undoubtedly, for some years to come, as the slit lamp increases in its number of users, we will be flooded with literature on corneal diseases, but eventually there will be a general classification of corneal diseases, based upon a more intimate knowledge of the processes that occur in the cornea as observed by the microscope aided by the slit lamp.

The same holds true of the lens. Only in recent years have we really appreciated the various conditions of the lens, thanks to the new methods of illumination introduced by Gullstrand, and it is to be anticipated that a great confusion in nomenclature of lens pathology will occur, until our knowledge is sufficiently widespread to justify more generalized classification than is at present possible.

One of the most interesting conditions to be observed by the slit lamp in connection with the lens occurs when the lens has been removed. We think with our modern methods we have obtained a beautiful result, but when we look with the slit lamp we find a junk heap worse than Yokohama—the lens body, capsule, cortex, pigment and what not. But these are microscopic and do not interfere with the vision.

I have been using an addition to the slit lamp which was made for me, in the form of a small glass disc ruled in millimeters. This disc slips into the central portion of Huygen's ocular. Naturally, this involves some confusion in the size required, so I have prepared a little table that I think you will find useful, and I am presenting it to you for future consideration.

Eye Pieces.

Objective	5x	7.5x	10x	12.5x
24	42x a .024	63x a .022	84x a .023	105x a .021
32	28x a .036	42x a .032	56x a .033	70x a .032
48	13x a .074	20x a .066	26x a .068	31x a .065

a = mm. represented by each division on scale.

x = diameter magnification.

One point that Dr. Bedell emphasized, is that the slit lamp is not a laboratory instrument; it is an instrument for routine clinical use. It

does not require a great deal of time, and you will find that with practice you will be able to use the slit lamp and lose very little time with each patient. The knowledge you will gain from the slit lamp more than compensates for the six to ten minutes you may have to spend in studying the lesions in question.

DR. ROBERT VON DER HEYDT, Chicago, Ill.: The introduction of microscopy of the living eye, made possible by the slit lamp, has been the greatest advance in ophthalmoscopic diagnosis of the past decade.

In connection with the discussion of the lens, I will endeavor to explain two practical instances where this method is also of value in prognosis. One can, for instance, tell at first glance as to whether a cataractous lens is in a state of threatening progression. This is the case if we find new fissures filled with fluid. These in their incipience are invisible by any other method of observation.

They appear like test tubes filled with a clear fluid within a denser media, and their presence prognosticate an impending progression. The spokes, when finally seen by focal light with the ophthalmoscope, are the end products of these fluid fissures, after the fluid has been absorbed. They are visible because the fissure walls are now permanently opaque.

The slit lamp also shows when a cataractous lens is ripe. This is shown by the presence of a subcapsular layer of flat vacuoles, seen by transillumination. They are in a layer of separation.

We may safely attempt to extract a lens in this stage, even without iridectomy, and are not in danger of stripping off a layer of soft cortex.

The slit lamp gives us very much that is new in our study of the anatomy of the lens. Dr. Blaauw has shown, that by means of it, we may see the normal lens in greater detail, because of the increased reflection given to its known and many newly found limiting surfaces.

Lens clouding, especially in its incipience, follows anatomic lines. Hence a study of these changes has exposed and made available these thus emphasized minute anatomic lines of demarcation.

As to Dr. Bedell's paper, I must say I am a little more skeptical as to our being able to differentiate the etiology by the character of the exudation within the anterior chamber in iridocyclitis. The same etiologic factor it seems will produce a greatly varied clinical anterior chamber appearance. We are just at the beginning in our study of these phenomena.

Regarding the dew like deposit on the cornea, Dr. Bedell stated it was difficult to differentiate between physiologic and pathologic dew like infiltration of the cornea. Physiologic dew is at the limbus, and represents an infiltration of the corneal epithelium, and possibly the stroma, by a nutrient fluid. All other dew like infiltrations of the cornea are absolutely pathologic.

DR. F. PARK LEWIS, Buffalo, N. Y.: Just a word in regard to nomenclature. I am wondering if, in the interest of accuracy, we cannot devise a phrase that will describe this instrument more specifically than that which is employed. Each one of the speakers this morning spoke of *seeing* with the slit lamp, which of course we do not do. They referred also to the corneal microscope. It is that no longer; it is now used not only for the cornea, but for the deeper structures. I am wondering whether some such name as "ophthalmomicroscope" would not better

describe the instrument, so that we may at least be accurate in nomenclature.

In regard to the first paper, I have seen this instrument used in Vogt's clinic, and I have not seen more accurate methods or more careful work there than that done by Dr. Blaauw, consequently I think the observations he has presented are thoroughly worthy of consideration.

One point, however, I wish to touch upon. The limitations of the instrument are those which any microscope must have. We see with it only three things in the eye: First, those that are opaque; second, those that are distorted, so that they give surface reflection; and third, those whose integrity is interrupted. With the best use of the instrument that we can make, that is all we can see in any eye, normal or abnormal. All that remains, the clear portion, we cannot see under any illumination. Therefore any study or record of the pathologic lens and of the internal tissues of the eye by means of the slit lamp is useful in determining its physiology, as Dr. Von der Heydt has suggested. Where we have opacities in the lens, either the lumen of the lymphatic channels has been obstructed, or the margin of these channels have become the seat of deposits, and in either event we have the beautiful figures that show the normal nutritive passages, which in a physiologic condition are absolutely invisible. By watching these changes as they take place, we are in the way of finding out the normal channels, which in no other way can we ever see.

DR. ALLEN GREENWOOD, Boston, Mass.: I would like to ask Doctor Blaauw if, in his observation of the crystalline lens, any marked changes are observed during the act of marked accommodation?

DR. ROBERT SCOTT LAMB, Washington, D. C.: I would like to ask Dr. Bedell if he has found any greater accuracy in his prognosis because of accurate observation?

DR. EDMOND E. BLAAUW, Buffalo, N. Y. (closing): In reply to Dr. Greenwood, no there is none. Not all lenses have the same thickness. I have not been able to study accommodation under the microscope.

I would say to Dr. Bedell that he will find a new field if he will stain his cornea with a 1 per cent sol. of methylen blue, preferably two years old. Fresh solutions are too irritating.

DR. ARTHUR J. BEDELL, Albany, N. Y. (closing): Answering Dr. Lamb's question regarding prognosis, by no other method can so much information be gained. Infiltrations are seen earlier, their progress noted, and the response to treatment observed.

Answering Dr. Von der Heydt, he will find in the paper that I particularly state that dew like changes are in the periphery, and I say that only at times is it difficult to differentiate, such as for instance in a limbus stippling, which is probably not infrequent in the earliest stages of interstitial keratitis.

TRACTS, OTHER THAN OTIC, FOR INTRACRANIAL INFECTION.

JOHN L. MYERS, M.D.

KANSAS CITY, MO.

The purpose of this paper is to refresh our memories relative to the tracts for intracranial infection, and to show some remote sources from which infection may pass into the brain and meninges, also to show various channels and plexuses of veins through which bacteria are carried into the cranial cavity. Further, we wish to arouse a deeper interest in studying, recording findings, performing autopsies and reporting cases of intracranial complications of other than otic origin.

In performing an autopsy, one is often surprised at finding disease within the skull, which arose from some apparently insignificant cause, located far away from familiar tracts leading to the brain. For instance, a man¹ working in a mine had a slight wound on his neck and arm. These became infected, and the patient died of brain complications. Autopsy revealed that the infection had been carried through the facial, angular, and ophthalmic veins into the cranium. A boy², six years old, was injured by a stick striking the left side of his face, making a wound midway between the angle of the orbit and the external auditory canal. The wound healed superficially, but later the child developed brain symptoms and died. Autopsy revealed a brain abscess in the left temporal lobe, caused by a pus pocket in the pterygoid plexus. A little girl³ falling, made a wound "at the root of the nose and to the left side". Infection was carried within the cranium and the patient died. "Autopsy revealed a cavernous sinus on the left side filled with pus, containing the same organism as previously found in the blood, with abscess in the cerebellopontine angle". Dr. Norton L. Wilson, who reported this case, says that he has seen seven of these cases. A man,⁴ forty years old, infected the right nares by picking it. Within a week he died of cavernous sinus thrombosis. Streptococcic sore throat⁵, peritonsillar abscess⁶, extraction of teeth⁷, a carbuncle on the side of the nose⁸, infected ethmoids, sphenoids, frontals and antra, postoperative infection in the tonsillar fossae⁹, operations on the septum, ethmoids and paranasal sinuses¹⁰, operations on the orbit—all of these have been the

cause of meningitis, brain abscess and sinus thrombosis. In reviewing the pathways and portals by which infection passes into the skull, our purpose is to be so concise that the most busy man, in a few minutes, may familiarize himself with them.

Every opening in the skull through which nerves make their exit or blood vessels pass, either from within out or from without in, is a potential pathway for infection to enter into the cranial cavity; however, there are some openings through which bacteria more readily and often pass. Of these we shall take notice.

By intracranial complication, we are to understand that certain conditions within the skull, as meningitis, brain abscess or sinus thrombosis, the origin of which is outside but introduced through any one of the natural openings, are to be considered. However, tuberculosis, syphilis and cerebral emboli due to cardiac disease, also brain lesions traceable to systemic sepsis are to be eliminated from this discussion; likewise, complications caused by a fracture of the skull, though this as an etiologic factor is demanding attention, because of the many cases of skulls fractured by auto accidents and the use of other modern machinery.

Because of infection being so frequently carried within through the auditory channels, the ear is recognized as the one single tract through which the greatest number of intracranial complications arise. Literature shows more cases of brain infection conducted through the otic channels, than through any other single portal of entrance.

In studying the pathways for intracranial infection, let us review the venous circulation both outside and inside the skull. The veins first observed are the frontal, supraorbital, angular, facial, temporal, internal maxillary, temporomaxillary, posterior auricular and occipital. The veins over the forehead and face are tributaries to the ophthalmic vein or some plexus of veins, and through these communicate with the sinuses within, whereas the veins back of the head are connected within through the parietal foramen, the mastoid foramen and the vertebral plexus, which is at the base of the skull.

Within the cranium, the venous blood flows between layers of the dura which are called sinuses. Of these there are seventeen in number: five single and six paired.

The single: Superior Longitudinal, Inferior Longitudinal, Straight, Circular, Transverse or Basilar.

The paired: Two Lateral, two Superior Petrosal, two In-

ferior Petrosal, two Cavernous, two Occipital, two Sphenoparietal.

The frontal, supraorbital and facial veins are tributaries to the ophthalmic vein, which enters the skull through the Sphenoidal Fissure and terminates in the cavernous sinus. Infection in the region drained by these vessels may often be conveyed by them into the cranium.

The internal maxillary and the temporomaxillary veins are connected with the:

Pterygoid Plexus

Location	Tributaries	Enters Brain	Connects with
Infratemporal and pterygoid fossae	Sphenopalatine V. Pharyngeal V. Infraorbital V. Posterior superior Alveolar V. Descending Palatine V. Buccinator V. Pterygoid V. Masseteric V. Inferior Alveolar V. Middle Meningeal V.	Through foramen ovale	1. Inferior Ophthalmic V. (anteriorly) 2. Anterior Facial V. (anteriorly) 3. Cavernous Sinus (superiorly) 4. Pharyngeal Plexus (posteriorly and medially) 5. Internal Maxillary V. (posteriorly)

Dental sepsis from either the inferior or superior maxilla, or infection about the palate or muscles of mastication may be carried to the brain and its membranes through the Foramen Ovale by this route.

We next consider the

Pharyngeal Venous Plexus

Location	Tributaries	Enters Brain	Connects with
Between constrictors and Buccopharyngeal fascia	Veins from tonsils Videan Vein Meningeal Vein	Carotid Canal Foramen Ovale via Pterygoid Plexus	1. Cavernous Sinus 2. Pterygoid Plexus (above) Internal Jugular (below)

Infection in and about the tonsils and the pharynx may be carried to the brain by way of the pharyngeal and pterygoid plexus, through the Foramen Ovale, and by way of the pharyngeal plexus through the Carotid Canal.

In the respiratory region of the nose is found the

Nasal Cavernous Venous Plexus¹¹

Location	Blood Supply from	Blood Flows	Blood Empties into
Over inferior turbinates and lower posterior part of septum	Sphenopalatine Artery	Forward	1. Anterior Facial V.
	Nasopalatine Artery		
	Anterior Ethmoidal Artery	Backward	2. Sphenopalatine V. to pterygoid Plexus
	Posterior Ethmoidal Artery		
	Nasal Branch External Maxillary Artery	Upward	3. Ethmoidal Veins into Cranium
	Septal Branch Superior Labial Artery		

"Zukerkandl¹¹ found that an anterior ethmoidal vein leads from the nasal mucosa and passes through the cribriform plate to and into the venous plexus of the olfactory bulb, or into one of the veins on the orbital aspect of the parietal lobe of the brain. These venous communications must be a factor in the intracranial complications that frequently accompany or follow some cases of inflammation of the nasal cavities and the paranasal sinuses." Dabney¹² shows that the cribriform plate of the ethmoid is the point of communication between the nose and the dural spaces, either by the venous or the lymphatic route, and by the fibers of the olfactory nerve. He collected nine cases of meningitis following submucous resection.

Let us now observe the anatomic regions, the channels through which infection may be conveyed and the portals of entry into the skull by which we may have

Thrombosis of the Cavernous Sinus

From	Through	Opening in Skull
1. Anterior Scalp Face Neck Orbit	1. Frontal, Supraorbital, Facial, Angular and Ophthalmic Veins	1. Sphenoidal Fissure
2. Orbit	2. Pterygoid Plexus Ophthalmic Veins	2. Foramen Ovale Sphenoidal Fissure
3. Nose	1. Ethmoidal Veins 2. Sphenopalatine Vein through Pterygoid Plexus 3. Anterior Facial, Angular and Ophthalmic Veins	1. Openings in Cribriform Plate 2. Foramen Ovale 3. Sphenoidal Fissure
4. Teeth	Pterygoid Plexus	Foramen Ovale
5. Tonsils	Pharyngeal Plexus and Pharyngeal and Pterygoid Plexus	1. Carotid Canal 2. Foramen Ovale
6. Neck and Occipital Region	Posterior Auricular, Occipital Veins, Vertebral Sinuses and Basilar Plexuses	1. Condylod Foramen 2. Foramen Magnum

In studying the reports of cases of intracranial complications due to infection about the forehead, the face, in the paranasal sinuses, the mouth and the pharynx, one cannot but observe and

mark the greater number of cases reported each year over previous years. This demonstrates that we are having more such cases, or else the profession is acquiring greater knowledge and becoming more proficient in detecting and diagnosing these conditions.

Frontal Sinus Infection as a Cause of Intracranial Complication¹³.

- 1896 Dreyfus collected 19 cases of intracranial infection due to the frontal sinus.
- 1908 Dreyfus collected 88 cases, and of these 36 were abscesses of the frontal lobe.
- 1914 Boenninghaus collected 87 cases of intracranial infection due to the frontal sinus.
- 1919 Gerber¹⁴ made a review of the literature and collected 240 cases of intracranial infection due to diseases of the accessory sinuses. 65 of these were brain abscesses.

Ethmoid Infection as a Cause of Intracranial Complication.

- 1903 Hajek¹⁵ asserted the cavernous sinus thrombosis was the classical form of intracranial complication for inflammation of the ethmoid labyrinth.

The Maxillary Sinus as a Cause.

- 1909 Hajek reported two cases of cerebral complication due to the maxillary sinus.
- 1914 Boenninghaus¹⁶ reported nine cases.

Sphenoid Sinus Infection as a Cause.

Boenninghaus collected 53 cases.

Cerebral Abscess	1
Meningitis	27
Cavernous Sinus Thrombosis	24

Dental Infection as a Cause.

Paunz¹⁷ reports one brain abscess of dental origin. Johns Hopkins Hospital¹⁸ reports three cavernous sinus thrombosis cases due to alveolar infection.

In a Report of Twelve¹⁸ Cases of Cavernous Sinus Thrombosis, the Focus of Infection was:

Paranasal Sinuses	3
Alveolar Infection	3
Facial Infection	2
Pharyngeal Infection (after Tonsil Operation).	1
Orbital Abscess	1
Not determined (probably sinuses).....	2

The portals through which infection from these cases may pass into the brain are the Sphenoidal Fissure, Foramen Ovale, Carotid Canal and the openings in the Cribriform Plate of the ethmoid bone. The other tracts that we wish to mention are the Foramen Magnum, the Parietal and Mastoid Foramen.

Within the cranium, anterior to the foramen magnum, is the basilar plexus, which is in the dura mater. It connects the posterior ends of the cavernous sinuses and the anterior ends of the inferior petrosal sinuses; also, it is connected outside the skull with the anterior spinal veins and the vertebral sinuses. The occipital vein¹⁹ communicates with the vertebral plexus, and is generally connected with the mastoid emissary vein, and its tributaries communicate with the parietal emissary vein. Therefore, an infection in the occipital vein may be carried to the longitudinal sinus²⁰ through the parietal foramen, the lateral sinus through the mastoid foramen, the inferior petrosal and the cavernous sinuses through the condyloid and foramen magnum.

How Infection Can Be Carried into the Brain

From	Through	To
1. Supraorbital, Angular, and Facial Veins with the Ophthalmic	Sphenoidal Fissure	Cavernous Sinus
2. Pterygoid Plexus	Foramen Ovale	Cavernous Sinus
3. Pharyngeal Plexus	1. Pterygoid Plexus to Foramen Ovale 2. Carotid Canal	Cavernous Sinus
4. Veins of Scalp	Parietal Foramen	Longitudinal Sinus
5. Posterior Auricular and Occipital Veins	Mastoid Foramen	Lateral Sinus
6. Vertebral Plexus	Foramen Magnum and Condyloid	Petrosal and Cavernous Sinuses
7. Nasal Plexus	1. Openings in Cribriform Plate 2. Foramen Caecum 3. Pterygoid Plexus 4. Facial Vein	Longitudinal Sinus Cavernous Sinus Frontal Lobe

BIBLIOGRAPHY.

1. Medical Journal Australia, November 12, 1921.
2. J. A. M. A., 1922, p. 1427.
3. Laryngoscope, 1921, p. 883.
4. Laryngoscope, 1921, p. 882.
5. Laryngoscope, 1921, p. 95.
6. J. A. M. A., September 17, 1921, p. 980.
7. Laryngoscope, 1921, p. 883.
8. Journal Laryn. Rhinol. and Otol., 1921, p. 173.
9. Archives of Surgery, November, 1920, 1, p. 483.
10. Annals Otol. Rhinol. and Laryng., June, 1923.
11. Schaeffer. The Nose and Olfactory Organ.
12. Surgery, Gynecology and Obstetrics, 1916, vol. 22, p. 328.

13. *Annals Otol., Rhinol. and Laryng.*, 1919, p. 113.
14. *Annals Otol., Rhinol. and Laryng.*, 1922, p. 493.
15. *Annals Otol., Rhinol. and Laryng.*, 1919, p. 140.
16. *Annals Otol., Rhinol. and Laryng.*, 1919, p. 141.
17. *Archives of Surgery*, November, 1920, 1, p. 483.
18. Cunningham. *Anatomy*, Fifth Edition, p. 974.
19. *Annals Otol., Rhinol. and Laryng.*, June, 1923

DISCUSSION.

DR. A. J. LORIE, Kansas City, Missouri: Knowing Dr. Myers' complete mastery of anatomy, I was not at all surprised to hear the fine details which are presented in this paper.

In my experience, I can remember three cases of this type of infection, which have occurred in the last few months. There is a peculiar and practical point in infections of the nose, which has been brought to my attention rather recently, and that is streptococcus infection of the columella and the upper portion of the upper lip; these cases almost universally are fatal, while infection in the alae and adjacent areas may get well if let alone. The point of infection in the vestibule of the nose is from the pernicious habit of pulling hairs.

The statement has often been made, that 95 per cent of intracranial complications develop from otitic sources. That has been due to carelessness, in a large proportion of cases, on the part of the profession in years past, in reporting complications of intracranial conditions due to intranasal sources.

Dr. Myers has shown the danger of operating upon acute conditions of the pharynx and nose, especially in attacking sinus conditions.

ORATION.

SOME CONCUSSION CHANGES MET WITH IN MILITARY PRACTICE.

SIR WILLIAM T. LISTER.

LONDON, ENG.

In seeking for a subject for my address, I naturally thought of work which I had done when I was one of your brothers in uniform if not in arms, and I have chosen certain of the concussion changes in the eye which, for the most part, are seldom seen apart from military practice.

With regard to the illustrations, I wish to acknowledge my indebtedness to the Committee of Medical Research for kindly allowing me to show some ophthalmoscopic drawings made for them by Mr. A. K. Maxwell under my supervision. Other ophthalmoscopic drawings and the colored lantern slides were made for me by Mr. A. W. Head. The photographs, both macroscopic and microscopic, together with the bulk of the lantern slides are my own.

I shall divide my remarks under the following headings:

- (1) Ruptures of the sclera.
- (2) Concussion changes of the iris and ciliary body.
- (3) Concussion changes in the retina and optic nerve.
- (4) Evulsion of the optic nerve.
- (5) The effects of foreign bodies striking the retina.

RUPTURE OF THE SCLERA.

I will take first ruptures of the sclera, a subject I dealt with in a lecture at the Royal College of Surgeons, the illustrations for which have never been published.

In civil life, it is well known that by far the greater number of ruptures of the sclera are concentric with the cornea, and take place either immediately above, or above and on the inner side, about 3 mm. from the limbus.

In military practice, on the other hand, though similar injuries may be seen, other, very different ruptures are met with; for example, the cornea may be burst forwards, or we find equatorial ruptures, or the whole sclera may be split into lobes.

In summarizing our experience of ruptures of the sclera,

we find that we have two main groups, according to whether the missile *has* or has *not* penetrated the globe.

We will first consider the *ruptures of the eye caused by a penetrating missile*.

The elasticity of the sclera will accommodate the entry of small particles without rupture. When, however, the fragment is of such a size and has such velocity that the united effect of the increase in volume caused by the entry of the body, together with the distending force of the pressure wave set up by its impact, is sufficient to overcome the elasticity of the supporting coat, widespread ruptures occur.



Fig. 1.

When a sufficiently large foreign body passes through the *back* of the eye, the cornea is shot forwards and the contents ejected; an effect comparable with that produced by firing a bullet through a canister full of water, when both the lid and the water are shot upwards. Fig. 1 shows an almost complete tearing away of the cornea due to a fragment passing through the posterior part of the globe and orbit.

When, however, such a fragment enters the *front* of the eye, there is an explosive effect, and the sclera is split into numerous lobes by radial ruptures which extend backwards from the point of entrance, in some instances as far as the optic nerve, so that when the sclera is removed, it resembles the calyx of a flower. I can show you two specimens illustrating this form of rupture:

Fig. 2 shows where a small particle passed right through the eye into the orbit. The sclera is seen split into numerous leaves, the lines of cleavage radiating out from the point of entrance.



Fig. 2.

Fig. 3 shows where two large splinters of wood were driven into the eye, splitting the sclera into two halves.

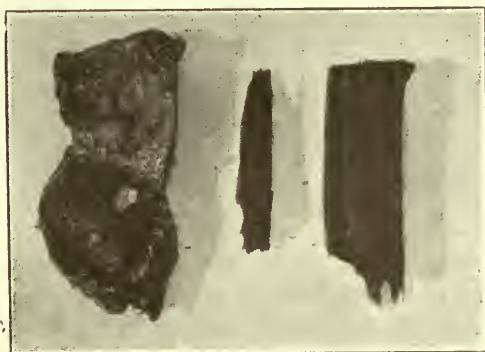


Fig. 3.

When the sclera is not perforated by the missile, the rupture does not start from the point of impact, but occurs at some considerable distance from it.

Rauber found, when investigating the effects of blows on hollow spheres, that the resistance of the envelope to pressure is one-third greater than its tensile strength. An eyeball would thus tend to burst at some distance from the point of impact

sooner than give way where the pressure is applied. In practice, we find that the site at which rupture takes place is most commonly either in the equatorial zone about the line of impact, or immediately opposite to the point of impact, (i.e. at the point of contrecoup), and that these two sites of rupture are associated with two different kinds of blow. (Fig. 4.)

(a) The ruptures caused by *slowly moving objects which strike the eyeball directly* without penetrating globe or orbit, occur in the equator around the line of impact, at a point where the globe is least supported. This group will include the bulk of ruptures met with in civil life, which are caused by the direct impact of objects such as stones, fists, boots, sticks, or cows' horns.

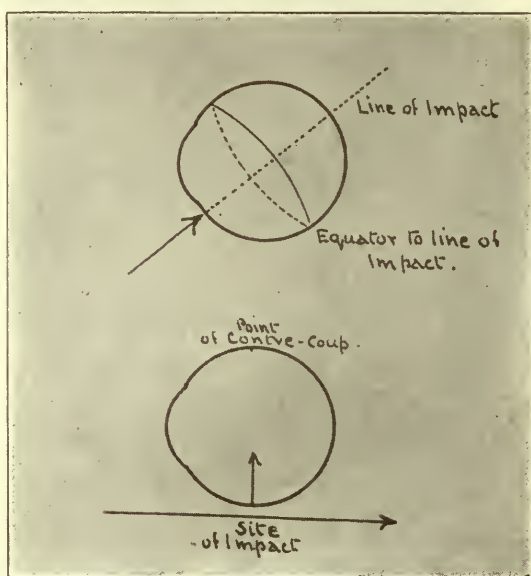


Fig. 4.

According to von Arlt's theory, indentation of the globe due to the impact of slowly moving missiles, by lessening the diameter in the line of impact, must necessarily cause an increase in the diameter in the equator round this line. Since a sphere has the greatest cubic content for a given surface, any force which distorts a sphere containing a practically incompressible fluid, must bring about an increase of surface—that is to say, the envelope will be distended. The eyeball, being supported both at the place of impact and at the pole opposite to this, distension will take place chiefly at the equator round the line of impact, and if the distension is sufficient to overcome the tensile cohesion of the

sclera, rupture will take place at some point on this equator where the globe is least supported.

The *site of impact* of such objects mentioned is at the anterior half of the eye, usually down and out, since protection is afforded to other parts of the eye by the brow, nose and cheek. The *line of impact* from a blow below and to the outer side of the cornea, will pass from below upwards and backwards and inwards; the least supported portion of the equator to that line of impact will be above and to the inner side of the cornea, and this is the place at which civil ruptures usually occur.

(b) In contrast to these we find that ruptures caused by *rapidly moving missiles which pass through the orbit but do not strike the eye directly*, occur at the side opposite to the point of



Fig. 5.

impact—i.e., at the point of contrecoup. For example, missiles passing through the back of the orbit cause the cornea to be burst forwards; or if they have passed through the side of the orbit, the rupture may be found near the equator on the opposite side.

I will now show some specimens illustrating these forms:

Fig. 5 shows rupture of cornea from passage of bullet at back of orbit.

Fig. 6 shows a similar condition due to the passage of a bullet through both orbits. Both optic nerves were divided by the missile; one at the optic foramen, the other close to the back of the globe. In the former the globe was whole; in the latter, though the back of the globe is intact, the sclera at the limbus is

ruptured and the cornea thrown forward. Both eyes were proptosed, projecting right in front of the lids. The patient was complaining of such intense pain that, much against one's inclination, the two eyes were removed.

Fig. 7 shows a large equatorial rupture on the inner side of the globe following the passage of a missile through the outer side of the orbit.



Fig. 6.



Fig. 7.

In these cases, the globe is not deformed by direct pressure, but is subjected to an indirect impact, or shock, due to the passage of the missile through the orbit. If the globe is not deformed, the equatorial zone will not be overstretched, and therefore von Arlt's theory will not afford an explanation.

I am greatly indebted to Dr. Love, Professor of Physics at

Oxford, who kindly considered the problem and has put forward the following explanation.

The contents of the globe being fluid, sudden impact at one spot on the surface will cause a wave of pressure to radiate through the vitreous from the point of impact (Fig. 8). The more rapid the impact, the more forceful the wave of pressure; the slower the impact, the less forceful the pressure wave.

The wave of pressure will be felt most by the sclera exactly opposite to the point of impact, where the wave strikes at right angles to the surface, while at the sides, where the wave strikes obliquely, the pressure will be less. Thus, it is familiar to us all that a wave of the sea which strikes a breakwater at right angles dashes much higher than one which strikes obliquely.

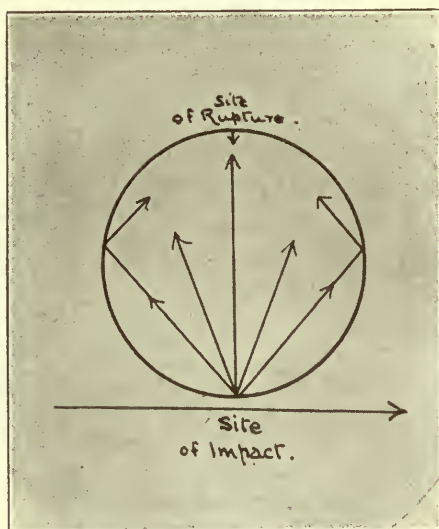


Fig. 8.

Again, where the incident wave strikes the sclera there will be a *wave of reflexion*, which will beat back against the incident wave. This wave of reflexion will only directly oppose the incident wave immediately opposite the place of impact; at other parts of the spherical surface, the wave of reflexion will be directed more or less away from the incident wave.

Professor Love, when calculating the effects of air waves, found that where the wave of reflexion met the incident wave the pressure is enhanced, it may be as much as eight fold.

Though one cannot insist too closely on the similarity of the effects produced by air waves and those caused by waves in the

semifluid vitreous, yet it would seem certain that a corresponding enhancement of pressure must take place in the eye at the point of meeting of the wave of reflexion and the incident wave.

Theoretically, the point of meeting of the incident and reflected waves will not lie exactly at the internal surface of the globe, but slightly nearer the center of the eye. This distance, however, will be infinitesimal, and consequently the enhanced pressure will be transmitted to, and borne by, the sclera immediately opposite the point of impact.

It is common knowledge with what force a sea wave reflected from a breakwater meets the oncoming wave. So great is the pressure here that a mass of water is shot up into the air. In this instance the wave of reflexion beats back against the succeeding wave, but in the eye the wave of reflexion will beat back against the incident wave. In the open, the breakwater does not bear the

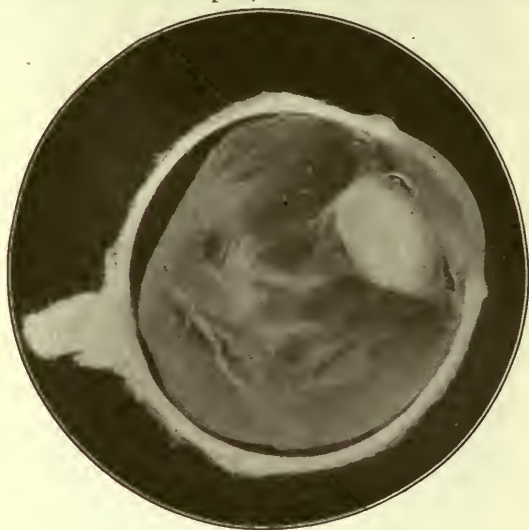


Fig. 9.

brunt of this enhancement of pressure, but in a closed space, such as the eye, this pressure must be borne by the walls.

It would therefore appear that the stress on the sclera produced by the incident wave, plus the enhancement of pressure due to the meeting of the incident and reflected waves, will be far greater at the point of contrecoup than at any other part of the globe, and thus ruptures at the point of contrecoup are explained.

CONCUSSION CHANGES OF THE IRIS AND CILIARY BODY.

Passing to concussion of the iris and ciliary body, I regret I have little original to say about these conditions, some of which

are met with fairly frequently as clinical pictures; we, however, seldom have reason for removing eyes with such injuries, and therefore rarely have the opportunity of examining the condition pathologically.

I have one specimen of iridodialysis (Fig. 9), which illustrates well the tearing away of the root of the iris from the ciliary body. Much more rare than iridodialysis,—with its consequent displacement of the iris centrally, giving rise to the D shaped pupil—is dislocation of the iris peripherally, which produces a pear shaped pupil in the slighter injuries or an “apparent iridectomy” in those more severe.

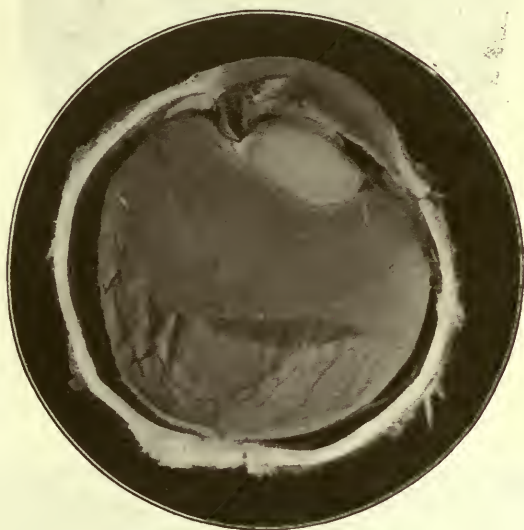


Fig. 10.

These “apparent iridectomies”, or peripheral dislocations of the iris, have features which distinguish them, as a rule, from true iridectomies. Thus (1), the pupillary margin, together with the pattern of the iris, passes right up to the coloboma, so that there are no cut angles of the sphincter to be seen; (2), there is no scleral scar; (3), no entanglement or prolapse of the iris; (4), the rest of the pupil remains central and circular; (5), there is a history of a blow, but no history of an operation. If, however, an iridectomy had been done, either the cut angles of the sphincter would be visible, or if these had been drawn up to the wound, there would usually be signs of entanglement or prolapse in a scleral scar, and the remaining portion of the pupil would not be circular, but drawn up toward the wound.



Fig. 11.

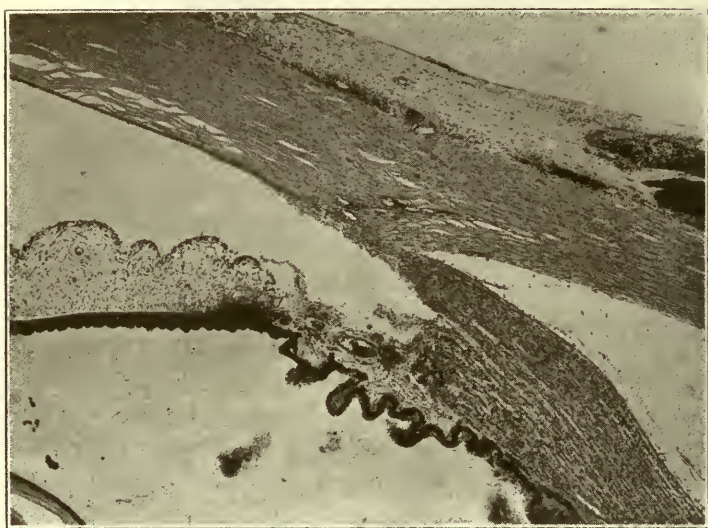


Fig. 12.

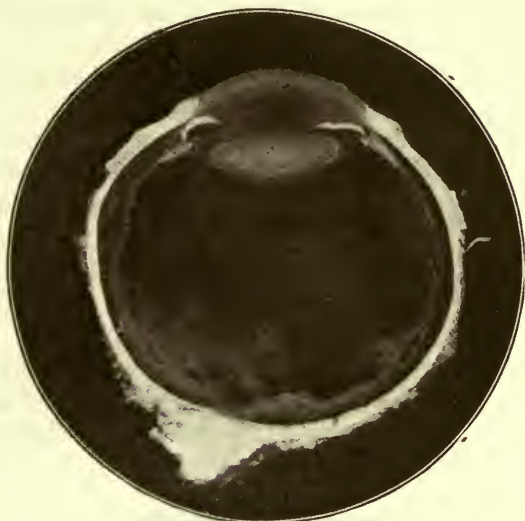


Fig. 13.



Fig. 14.

Such cases of "apparent iridectomy", or peripheral dislocation of the iris, are accounted for either by:

(1) Retroflexion of the iris, i.e. the iris is bent back and caught between the ciliary body and lens, (Fig. 10,) or by:

(2) A rent in the ciliary body—

(a) In some, the rent is in the substance of the ciliary body, separating the circular from the longitudinal fibers. (Figs. 11 and 12.)

(b) In others, the rent occurs at the junction of the ciliary body with the sclera, viz., a "cyclodialysis". (Figs. 13 and 14.)

In either case, the iris with the circular fibers alone, or with the whole thickness of the ciliary body, circular and longitudinal fibers together, slide outwards towards the equator of the eye. According to the extent of the displacement, so is the clinical

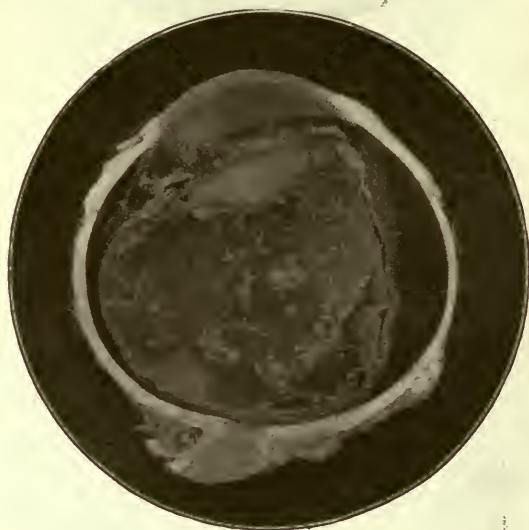


Fig. 15.

picture. If the displacement is less than the width of the iris, a pear shaped pupil results; if, however, the displacement is as great as, or more than the width of the iris, a coloboma occurs. In very rare cases (Fig. 15.), the cyclodialysis is complete, and the whole diaphragm formed by the lens, suspensory ligament, iris and ciliary body is dislocated backwards.

The usual explanation of these cases seems quite sufficient. Thus, when the cornea is indented by the missile, the contents of the eye being fluid and incompressible, a compensatory enlargement of the posterior segment of the eye must take place.

The pressure of the aqueous forces back the iris against the lens and, therefore, no fluid can pass through the pupil, with the result that the iris, lens or both must recede, or something must give way.

When the pressure is sufficient, the fluid wedge of aqueous humour may bring about any of the above mentioned conditions, namely iridodialysis, retroflexion of the iris, splitting of the ciliary body, or partial or complete cyclodialysis.

CONCUSSION CHANGES IN THE RETINA AND CHOROID.

In civil life, the changes are, for the most part, due to blows from comparatively slowly moving objects, which strike the anterior part of the eye directly. These blows cause disturbances located chiefly in the macular and perimacular region: viz., commotio retinae, detachment of the retina, choroidal ruptures, pigmentary changes in the macula, retinal hemorrhages and retinal rents, with which we are all familiar.

In military practice, similar changes caused by blows from stones, clods of earth etc. were met with; but in addition, concussion changes were seen caused by rapidly moving missiles, which passed through the orbit without rupturing the sclera, concussing the eye equatorially or in the region of the posterior pole. They gave rise, when examined soon after injury, and when the vitreous was not clouded by hemorrhage, to one of the most dramatic appearances the ophthalmoscope reveals. We saw great blood red clouds of hemorrhage in the retina interspersed with glistening white areas.

If examined some weeks later, a great alteration was found to have taken place. The glistening white areas had vanished; the hemorrhages, for the most part, had disappeared or were in process of absorption, or they had been replaced by fibrous tissue plaques in the substance of the retina or in the vitreous. These plaques often had festooned margins, and at times appeared perforated by round or oval holes. The probable explanation of the festooned edging is, that the plaques had been at one time larger, and were attached to the retina at certain spots on their periphery: as contraction took place, the outline became bayed between the fixed points. It is more difficult to account for the holes in the plaques, but on the supposition that complete absorption takes place at a certain spot in the plaque, retraction of the fibrous tissue will leave an oval or circular hole.

These concussion changes in the retina and choroid occurred in three situations:—

(1) The area always affected was that adjacent to the site of impact. So constant was this, that in cases in which the entrance wound was insignificant, the ophthalmoscope gave the first means of information that a foreign body had traversed the orbit.

(2) The next most common site to be injured was the macular region, and it is to be noted that the disturbed area at the macula was often quite separate from that adjacent to the site of impact. It is probable that the greater vascularity and delicacy of structure of this region rendered it more vulnerable to the intraocular waves of pressure.

(3) In a few cases, hemorrhages were also found opposite the site of impact. Professor Love's theory of the rupture of the sclera at the point of contrecoup explains such lesions, and if I am allowed to digress for a moment, it also explains how it is

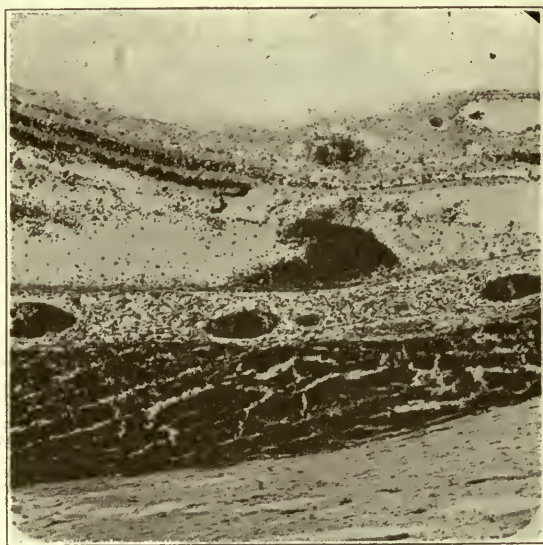


Fig. 16.

that severe blows on the skull cause bruising of the brain at the pole opposite to that which is struck.

Turning to the histologic changes found in these injuries, I regret I am unable to show you any specimens demonstrating the changes in commotio retinae. It is, however, known that in concussion of the brain, imbibition of fluid takes place in the colloidal brain matter; it is attractive to suppose that the retina, being part of the brain, may also imbibe fluid after concussion, giving rise to the appearance commonly described as "edema of the retina".

The hemorrhages in and about the retina and choroid are of every variety, subhyaloid, interretinal and subchoroidal.

The four main changes found in the retina are:

(1) The shrinking and disappearance of the nuclei of the granular layers.

(2) Vacuolation of its substance.

(3) The splitting of the retina into layers.

(4) Folding of the retina.

The degree of changes in the retina varies greatly in different specimens, and also in different parts of the same specimen.

In the less affected parts, we may find merely a partial and patchy atrophy of the nuclei of the external and internal granular layers, demonstrated by a diminution in size, so that these vary from the normal to minute dots. (Figs. 16 and 17.)

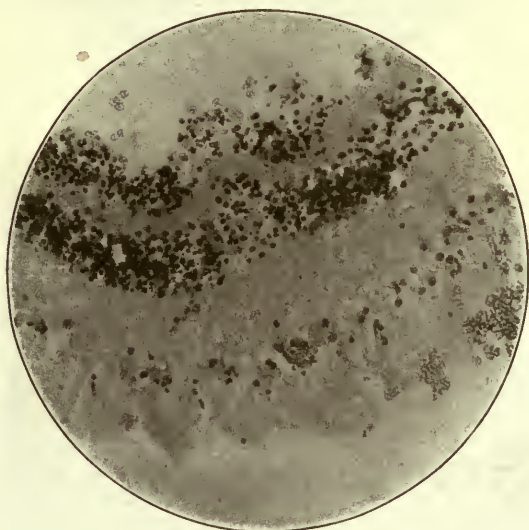


Fig. 17.

(This is comparable with the shrinking of the nuclei seen in atrophic areas of glioma of the retina). With the shrinking and disappearance of the nuclei, the retina loses its staining capacity, so that one finds all stages from a diminution in the size of the nuclei, occurring in patches, to a practically nonstaining band in which all traces of nuclei have disappeared.

Associated with the atrophy in the granular layers, the nerve fiber, ganglion and reticular layers also undergo changes. They become spongy, vacuolated and in places cystic. (Fig. 18.)

In the more severe cases the retina may be found split by hemorrhages into several layers (Fig. 19).



Fig. 18.



Fig. 19.

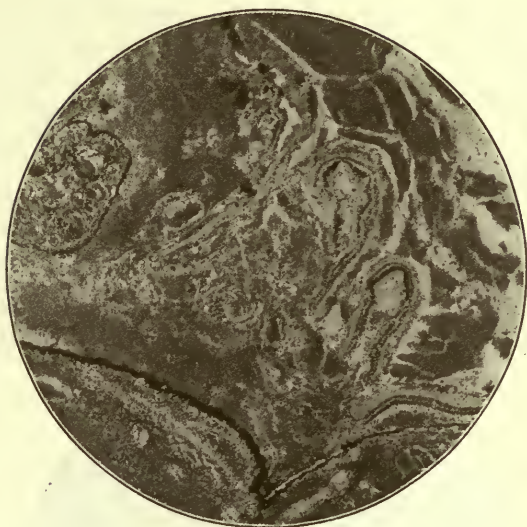


Fig. 20.



Fig. 21.

Every variety of wrinkling of the retina occurs; it may be fairly flat or it may be thrown into complicated convolutions. The combined effect of necrosis with splitting and folding of the retina gives some of these sections a remarkable and characteristic appearance, which may be compared to a tangled skein, or a ghost like retina wriggling through a mass of blood clot. (Figs. 20 and 21.)

What are these atrophic changes in the retina due to? In all the specimens examined, the blood vessels were severely damaged and, therefore, the nutrition of the retina must have been greatly impaired. Is the cutting off of the blood supply the only cause of the atrophy, or is there some other factor? It is very striking.

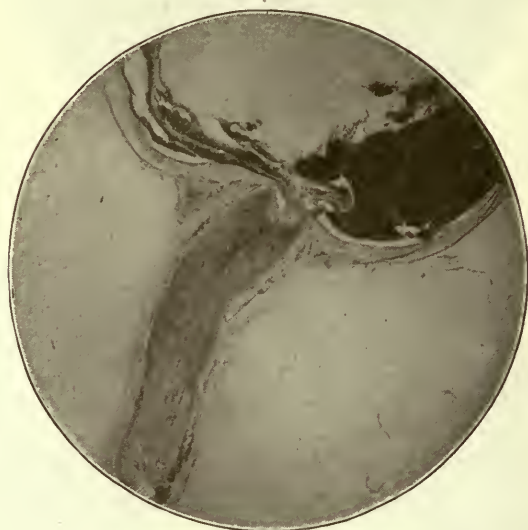


Fig. 22.

that in certain specimens where the blood supply was completely cut off by rupture of the arteria centralis retinae and the short ciliary arteries, the atrophy of the retina was not universal but only partial.

Given sufficient time, the cutting off of the blood supply would of course cause atrophy of the retina, which one would expect to be uniform. In the cases mentioned, it would seem, therefore, that there was some other causative factor for necrosis in addition to the cutting off of the blood supply. What can this other injurious factor be? The only suggestion which appears to me reasonable, is that the concussion brought about sudden death of certain parts by direct application of force. Such a view is well supported by the investigation of concussion of the spinal cord

by Gordon Holmes, which he described in his Gulstonian Lectures. He showed that areas of atrophy occurred in the cord, after the passage of a missile in the immediate vicinity, apart from any hemorrhage or visible vascular change, and he attributes such atrophy to the effect of direct concussion.

Though I have no specimen of concussion of the cord, I have a case of division of the optic nerve by a missile, which demonstrates the loss of staining power at the site of the lesion and the vacuolation and absence of nerve structure in this region. (Figs. 22 and 23.)

From what we have seen it appears evident that the concussed retina is an *atrophic* retina, associated with varying degrees of



Fig. 23.

corrugation, but we have yet to correlate the ophthalmoscopic with the histologic appearance, and to try to determine the nature of the glistening white areas. Here we meet with a difficulty. On the one hand, the concussed eyes which could be profitably examined with the ophthalmoscope, we had as a rule no justification to remove, and we could not, therefore, cut sections of a given area of retina in which the white spots had been seen. On the other, in severely concussed eyes which were both blind and painful, and which were excised, careful search failed to discover any white spots. What had happened to them? Had absorption taken place before the excision of the eye of some substance which gave rise to the white appearance, or had the forma-

lin, which was the fixative used, dissolved this material and rendered the retina transparent?

I am unable to answer these questions with certainty, but I should like to make a comparison which I think may throw light on the subject. In embolism of the central artery, the retina is white and undergoing coagulation necrosis. In time, the white appearance disappears as the retina passes into complete atrophy, just as the white appearance disappears in a concussed eye. Though the glistening patches in a concussed eye appear whiter than the retina whose artery has been blocked, this effect is probably due to contrast, for while in embolism hemorrhages are uncommon, and there is nothing dark in the fundus to show up the white patches, in concussed eyes the white areas are frequently surrounded by extensive dark red hemorrhages.

In the absence of direct proof, it thus seems a likely hypothesis, that the glistening white areas in concussed eyes are areas of retina in which coagulation necrosis has taken place.

EVULSION OF THE OPTIC NERVE.

Among other injuries of the optic nerve, I will say a few words about evulsion, and show some of the specimens which Montague Hine and I described at the Meeting of the Ophthalmological Society of the United Kingdom in 1919.

Looking back at the various ophthalmoscopic pictures which have been shown and published, to which the term "evulsion of the nerve" has been applied, I suspect that a considerable number may have been illustrations of "division" of the nerve without "evulsion".

The special feature of many of the drawings labelled "evulsion of the nerve" is a mass of fibrous tissue covering the disc, and a disappearance of the blood columns in the retinal vessels.

But an anterior division of the nerve will cause similar severe concussion changes in the retina, with formation of fibrous 'issue which may cover the head of the nerve, and provided the lesion is sufficiently far forward to stop the circulation in the central retinal vessels, an identical clinical picture will be produced. It is impossible to determine from these pictures the crucial point, namely, whether the lamina cribrosa is intact or not.

It is probable that only in those cases where the optic disc is unobscured by hemorrhage or fibrous tissue, and a definite pit is seen at the site of the nervehead, can one state with certainty that evulsion has taken place.

I have the ophthalmoscopic picture of only one case in which

evulsion was diagnosed before removal, and proved to be present by pathologic investigation. (For Figs. see Trans. Oph. Soc. U. K., 1919.) It is one in which a flake of metal, 14 mm. long, 10 mm. wide, and 4.5 mm. thick, had entered at the inner end of the right upper lid, traversed the nose and passed to the back of the left orbit, coming to rest at the posterior third of its outer wall. The right eye was completely ruptured and had been removed. The left eye was blind, but showed no external signs of injury; the ophthalmoscope, however, revealed a sharply punched out hole in the position of the optic disc, surrounded by retinal hemorrhages and other concussion changes. No definite retinal blood vessels could be seen, but there were a few red streaks just above the disc, and it was difficult to decide whether these were attenuated blood columns in remaining retinal vessels or blood streaks in the vitreous.

The patient subsequently died from septic meningitis, the result of penetration of the brain by other foreign bodies, and thus an opportunity of pathologic investigation both of the eye and orbit was rendered possible.

It was found that the optic nerve was still attached to the eye and had not been divided, but its sheath had been cut on one side only, either by the missile itself, or possibly by a fragment of bone which had been driven across the orbit. The nerve tissue, together with the lamina cribrosa, was pulled out from the globe, and its end was seen lying in the gap of the nerve sheath. The sheath was filled with organising vitreous, and there was no empty sleeve as would have been expected from the ophthalmoscopic picture, but as the drawing was made some days before death, it is probable that it had been filled with this new formed tissue in the last few days of life.

Though our experience of clinical cases was so small, the routine examination of blind, painful eyes which had been removed yielded us some beautiful specimens of this condition, which were unsuspected before removal, since ophthalmoscopic examination was rendered impossible by intraocular hemorrhage.

CASE 1. The missile had cut through the whole nerve close to the globe, so that at the time of excision, as soon as the conjunctiva and muscles were divided the eye came away. There was gross concussion of the retina and choroid, together with infection of the eye, which had probably spread forwards from the track of the missile through the opening in the nerve. In this case there was *partial evulsion* of the nerve tissue, the central fibers only being absent, the peripheral parts of the nerve remain-

ing in the sheath. This specimen supplies the anatomic counterpart of those cases where there is a hole occupying *part* of the disc.

CASE 2. Showed the nerve sheath entirely devoid of any nerve tissue, and containing merely a process of vitreous projecting backward into the empty sleeve; the retina, which showed gross concussion changes, had been torn in a ring about $1\frac{1}{2}$ mm. from the margin of the disc, while in front it had also been torn away from the ora serrata almost all the way round.

CASE 3. Showed also a nerve sheath completely devoid of any nerve tissue or trace of lamina cribrosa.

CASE 4. The specimen showed a huge hemorrhage which occupied the posterior half of the eye and extended into the nerve sheath. Some coiled remains of retina, which had been dragged back when the nerve was retracted, were seen also partially occupying the sheath.

Except in the case of partial evulsion where the whole nerve was cut by the missile close to the globe, the specimens showed a tearing out of the nerve tissue without any separation of the nerve sheath from the back of the globe.

In considering the etiology of evulsion we find that it follows:

- (1) Blows on the front of the eye.
- (2) Penetrating wounds of the ball itself.
- (3) Penetrating wounds of the orbit, either at the side or the back of the globe.

The actual mechanism of evulsion will vary with the nature of the injury. The nerve must be either pushed out or pulled out.

For evulsion to take place after a blow on the front of the eye or after penetration of a foreign body, or again after the lateral impact from missiles passing at the side of the orbit, the only mechanism that one can suggest is that the increase of intra-ocular pressure, which the injury produces, is sufficient to break the lamina cribrosa and expel the nerve. Hence such cases would be more correctly described as "expulsion" rather than "evulsion" of the nerve.

Though one might expect that rupture of the sclera would take place in one of the ways already mentioned in Section (I) more readily than a pushing back of the lamina cribrosa, yet the fact remains the lamina is found broken through. In seeking for an explanation, it must be remembered that the lamina is very weak and at most represents only one third of the fibers of the sclera, and also that the nerve itself is only slenderly attached

to its sheath. When once, therefore, the transverse fibers of the lamina have given way, there will be little resistance to expulsion of the nerve, and the appearance of the empty sleeve would be accounted for.

On the other hand, the nerve can be pulled out in one of two ways, either by the explosive effect of a missile passing through the back of the orbit, or by extreme rotation of the eye; in both instances the optic nerve will be severely stretched.

Dr. Turnbull kindly prepared sections of the optic nerve and sheath, staining them to demonstrate elastic fibers. These conclusively show, that while the sheath contains elastic tissue, the nerve contains none. The sheath is thus more elastic than the nerve. The explosive effect of a missile passing through the back of the orbit will have the double effect of forcing the eyeball forwards, and thus stretching the nerve, and at the same time pushing the nerve out of its course, putting it still further on the stretch. If the sheath is stretched, its contained nerve tissue, being less elastic, will tend to break rather than stretch. Why should the nerve break at its junction with the retina, rupturing the lamina cribrosa, and be pulled back like a piston in a cylinder, instead of breaking in its continuity? This may be explained by the fact, that between the lamina and the retina, the nerve tissue consists almost entirely of naked axis cylinders, whereas behind the lamina, the nerve has strong supporting fibrous lamellae which are continuous with the lamina. The nerve is, therefore, tougher behind the lamina than in front. If the nerve sheath is stretched, the toughened but inelastic nerve, sliding easily in its dural coat, tugs at the lamina, and when once this gives way, a rupture will tend to take place in front of this membrane, either in the retina itself or in the nerve fibers between the retina and the lamina. This also explains why we sometimes find a hole torn in the retina close to the disc, and in others a tangle of degenerated retina dragged into the open mouth of the sheath.

Lastly, we have to explain how extreme rotation of the eye from blows with sticks or poles may cause evulsion. Extreme rotation will cause tension on the nerve where it enters the globe, and especially on the side away from which the eye is rotated. If such tension be sufficient, rupture of the lamina cribrosa and evulsion of the nerve may take place, either complete or partial, for the elastic sheath will stretch, but the inelastic nerve will pull on the lamina and cause it to rupture (cp. Lang's and Hesse's cases).

THE EFFECTS OF FOREIGN BODIES STRIKING THE RETINA.

I will conclude by showing you a few specimens which demonstrate the effect on the internal structure of the eye of penetrating foreign bodies.

When a foreign body passes through the vitreous, it may leave a track which may be either filled by hemorrhage, or subsequently marked out by a fibrous band. The larger fibrous bands, by their contraction, may cause severe disorganization, such as detachment of the retina or choroid or both, or general distortion of the globe.

When a foreign body strikes the retina, a variety of things may happen. It may remain embedded in the retina, in which case it often causes puckering of this membrane (Pl. I and Fig. 25); or if it strikes the back of the eye with sufficient force it may penetrate its coats and pass into the orbit.

It is not infrequent to find that a foreign body, having struck the retina, may rebound and come to rest far from its original track. We saw one case of which the only explanation I can offer is that a fragment rebounded twice on the retina. There was a slightly pigmented scar in the sclera at the entrance of the foreign body on the inner side of the cornea. The ophthalmoscopic picture showed two scars on the retina, one on the inner, the other on the outer side of the disc. These were connected by a fan of fibrous tissue, which could be traced forwards towards the wound of entrance. The foreign body was seen like a jewel in the blood stained vitreous, situated far forwards and on the outer side. An X-ray photograph demonstrated the foreign body at a spot which corresponded with the ophthalmoscopic estimation, and there was no indication of any other foreign body in the orbit. The field of vision showed two scotomata situated in the horizontal meridian corresponding with the two scars on the fundus.

When a foreign body strikes the retina, it will certainly bruise it and in most instances cut it. I have sections of a case in which a foreign body bounced on the retina and was later removed by means of Haab's magnet. The spot where the foreign body struck the retina was indicated by the attachment of the vitreous. The sections through the spot (Fig. 24) showed the retina to be divided, and the gap thus caused was occupied by a plug of newly formed fibrous tissue, which was continuous with the shrunken and hemorrhagic vitreous, in which fibrous tissue was beginning to replace the blood.

Following these minute injuries of the retina in eyes which had not been too severely disorganized, we were at times able to correlate the visible lesions with the corresponding defects in the field.

Thus we found that lesions above or below the horizontal plane caused a defect in the field out of all proportion to the local disturbance, i.e., there was a "distribution defect" in addition to the local defect due to the lesion. This was due to the foreign body having not only damaged the rods and cones at the



Fig. 24

spot which it struck, but to its having there divided the nerve fibers which were passing on to a more peripheral portion of the retina. The distribution defect was fan shaped from the point corresponding to the lesion, and extended towards the periphery or the median raphe. The nearer the lesion was to the disc, the greater the blind sector and vice versa, as would be expected from the accepted distribution of the nerve fibers in the retina.

When a lesion occurred in the horizontal line drawn through the disc, there was no "distribution" defect, but the loss of field was limited to the area corresponding with the lesion, as would

be expected, for the median horizontal raphe is the terminus for all nerve fibers on this line.

These sector defects following injury of the retina are strikingly similar to those following solitary patches of choroiditis, in which the resulting blind area is best explained on the supposition, that the nerve fiber layer is involved and destroyed by the inflammatory process as it passes over the patch of choroiditis. This supposition is supported by microscopic examination of a patch of choroidal atrophy, which frequently shows complete disorganisation of all layers of the retina, together with the choroid.

The last case I wish to bring to your notice, is one in which a minute particle perforated the cornea, iris, lens and vitreous, and struck the optic disc a "bullseye". There was an adherent leucoma, a hole in the iris, and an opaque streak through the lens, from which there extended a delicate fibrous band, through the vitreous, to the mass of inflammatory tissue covering the disc and the parts immediately around (Pl. II). An X-ray photograph proved the presence of a foreign body situated on the disc; the rest of the fundus was normal. The astonishing fact is that this lesion caused a nearly circular "ring" field. That is to say there was a very large central scotoma, a considerable peripheral contraction of the field, and between the two a nearly circular band in which vision was retained (Fig. 26). In the absence of histologic investigation of the nervehead, one can only guess the exact portion of the nerve which had undergone degeneration, and how the foreign body caused the circumscribed injury, whether by direct impact or by subsequent degeneration following the injury.

According to the generally accepted views regarding the distribution of the nerve fibers, those in the outer third of the disc supply the macular area, the most peripheral fibers of the disc supply the retina in its immediate neighborhood, while those in the center are the long distance fibers, which supply the periphery of the retina. Between these three sets of fibers there would be a horseshoe shaped zone on the disc, the fibers from which would pass to the portion of retina corresponding with the above mentioned ring field. It would hardly appear creditable that a complete "ring" field could have been left, yet it has to be accepted as fact, since the field was taken most carefully on two separate occasions, at an interval of several weeks, and the two fields were practically identical.

The important fact which emerges from this case, is that *a wound of the disc led to a double concentric defect of the field.*

The case thus falls into line with certain cases of optic nerve disease such as are met with occasionally associated with Ring Scotoma, e. g., Tabetic Atrophy, and Retrobulbar Neuritis, and it also gives support to the possible view that retinitis pigmentosa, with its concentrically defective fields, may be primarily a disease of the nerve.

Lastly, it appears to prove that the nerve fibers which are in association with the various concentric zones of the retina, and which will be distributed to the corresponding zones in the appropriate superior and inferior calcarine areas of the occipital lobes, are arranged in definite zones in the optic nerve.

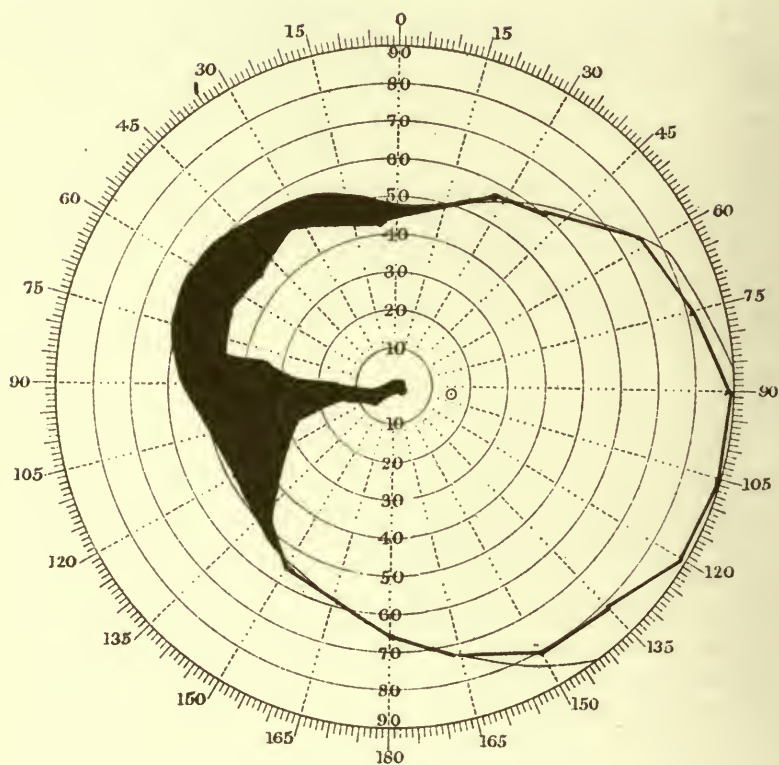


Fig. 25. See also Pl. I.



Pl. I. Ophthalmoscopic picture showing puckering of the retina due to a penetrating F. B. To the oval opalescent swelling is attached a band of fibrous tissue in the vitreous which indicates the track of the F. B. See also Fig. 25.

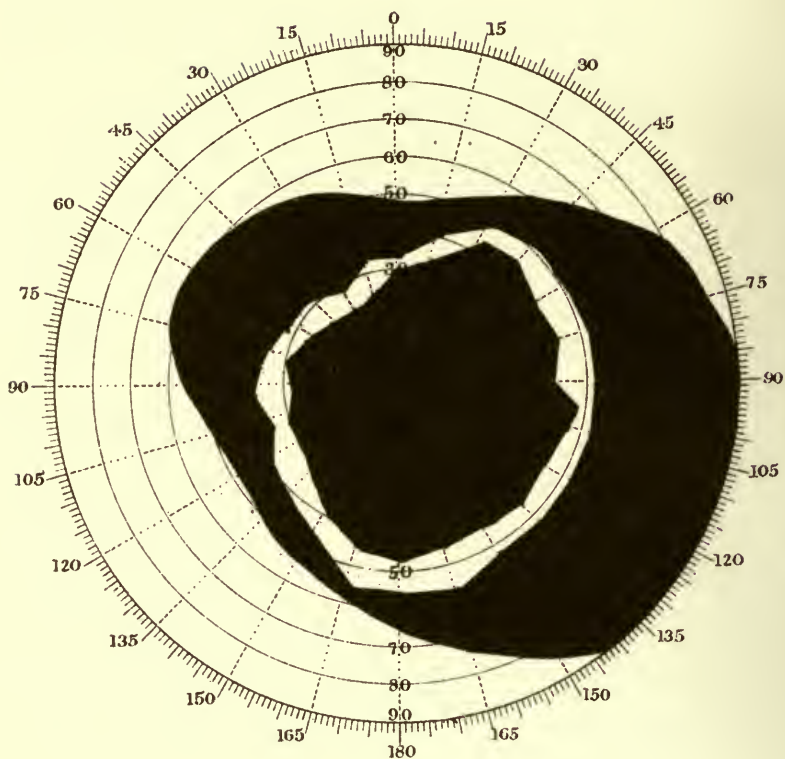
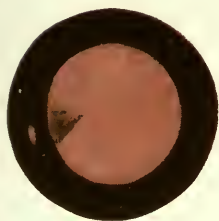


Fig. 26.

Fig. 26. The field of vision shows a large central scotoma and peripheral contraction, between which has been left a nearly circular ring field. See also PL II.



Pl. II. Drawing showing the course of a penetrating foreign body which passed through the cornea, iris, lens, vitreous and came to rest at the optic disc.

There was an adherent leucoma, a hole in the iris, an opaque track through the lens, a fibrous band passing from the back of the lens to the optic disc.

The disc is covered by a mass of fibrous tissue through which the retinal vessels emerge. See also Fig. 26.



THE OCCURRENCE OF CARTILAGE IN THE TONSIL.

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From the standpoint of the practical clinician, the presence of cartilage in the tonsil is more or less of an academic curiosity, which has little bearing on the every day work of the laryngologist. It is, therefore, all the more to the credit of the colleagues practicing this specialty, that this and kindred themes are topics for consideration in their scientific gatherings, evidencing a love for pure research without utilitarian motives.

Of late, quite a number of papers have appeared in medical literature on this subject. The increased interest is probably due to the accidental discovery of islands of cartilage in the tonsil in the course of routine examinations which are conducted nowadays in modern hospitals, where, in accordance with the standardization requirements, all tissues removed at operations are sent to the laboratory for macroscopic and microscopic observation. The discovery of cartilage or bone in such an unusual location strikes the pathologist as something out of the ordinary, but he gets little enlightenment from the surgeon who has removed the tonsils, as the text books on diseases of nose and throat make no mention or give but meager reference to these findings. Elated over this novel discovery, he is chagrined in looking over the periodical literature to verify the correctness of the verse in Ecclesiastes, that "There is nothing new under the sun." True, the first report of the occurrence of cartilage in tonsil dates back to recent times, still within the memory of the present generation. The classic reference to bone and cartilage formation in the tonsil is found in a contribution to the Jubilee Festschrift, commemorating the fiftieth year of medical activity of Rudolph Virchow. The paper in question is from the pen of no less an authority than Professor Johannes Orth, who occupied the chair of Pathology in the University of Berlin for many years. It is a brief description of the occurrences of bone in the tonsils in a man, aged 59, and of cartilage in a two year old child. In view of the apparent absence of inflammatory changes, Orth ascribes their presence to congenital disturbances, such as rests of the branchial arches in the neighborhood of which the tonsils develop.

Two years later, his pupil, H. Deichert, writing in Virchow's Archiv, describes similar findings in three cases, with an extended report of the histologic characteristics and an elaborate discussion of the origin, which he also attributes to congenital anomalies. In 1898, the British Lancet published two articles on the occurrence of cartilaginous and bony nodules in the tonsil by Walsham and by Wingrave, respectively, who followed the preceding investigators in their interpretation of these findings as a congenital aberration.

At the beginning of this century, the question of metaplasia or change of one kind of tissue into an allied form occupied the attention of pathologists. Lubarsch as well as Pollack, in their studies on ossification in old tuberculous lymph nodes and calcified tuberculous foci in the lungs and pleura, attributed the process to metaplasia. Pollack's work includes a consideration of cartilage and bone of the tonsils, in which organs he found evidences of previous inflammatory activity, which he interpreted as examples of metaplasia. In accord with the same explanation, appeared a publication by Nösske in 1903, who reported six cases of cartilage and bone in the tonsils, and supports his thesis in favor of metaplasia as against the embryonic theory by a formidable argument. While, heretofore, cartilage and bone in the tonsil had been considered a rare find, Nösske hazards the opinion that it is far more frequent than is supposed.

In the following year, Ruckert in Virchow's Archiv, reporting on examination of tonsils of adults and newborn, takes up the cudgel in behalf of the congenital aberration theory. Since then, a number of other papers have appeared in the literature at intervals, the most recent being the valuable scientific contribution of Dr. Weller of the University of Michigan, which was published in the Annals of Laryngology while our modest effort was in course of completion.

In view of the controversy between the two schools as to the explanation of the origin of cartilage and bone in this situation, a dispute which has not yet abated, it may not be amiss to give a brief summary of the arguments advanced by both sides, if for no other reason than to recall to the clinicians here present some long forgotten lore from the domain of pathology and embryology.

The "Anlagen" of the tonsils appears in early fetal life. They are visible in the fourth month, at first as simple invaginations of the mucous membrane at a point between the second and third branchial arches, i.e., at the second branchial pouch.

If we take the Eustachian tube, derived from the first branchial cleft, as an analogous example, islands of cartilage are found in the connective tissue bands joining the tube and the pharynx (ligamentum salpingopharyngeale). From the second branchial arch are developed such structures as the styloid process, stylohyoid ligament, styloglossus muscle and others, in which aberrant islands of cartilage are occasionally found. All these are contiguous to the tonsil. How plausible, then, is the assumption, that in the further development of the structures of the pharynx, accompanied by relative changes in position, portions of cartilage may at times be left at the periphery of the tonsil and continue their growth. The explanation is further strengthened by the occurrence of cartilage in tonsils of the newborn, where inflammatory changes can have played no role. This view is still reflected in some of the text books on pathology, notably that of Ribbert, as well as in the last edition of Aschoff, both of which authors, in their brief reference to the anomalous occurrence of cartilage and bone in the tonsil, assign embryonal misplacements from the second branchial arch as the cause.

The school which considers cartilage and bone in the tonsil as the result of metaplasia, cites examples of similar processes in other parts of the body in support of their argument. During the process of regeneration, cells of kindred origin may change into one another as the result of altered external conditions. Thus, in exstrophy of the bladder, the transitional epithelium changes into the squamous variety, with formation of a horny layer. Similarly, in prolapse of the uterus, the cylindrical cells are replaced by a stratified squamous layer. The same holds true of cells of the connective tissue type. Just as we find fibrous tissue replaced by adipose or mucoid tissue, so there may ensue a transformation into cartilage or bone. Thus we encounter bone in the walls of arteries, in the valves of the heart, in calcified necrotic tissue, such as tuberculous lymph nodes and lungs. In all of these metamorphoses, certain limits cannot be overstepped. In the higher animals, Virchow's famous dictum "*omnis cellula e cellula ejusdem generis*" still holds true. Connective tissue cannot turn into epithelial or vice versa. It is only the various types of connective and epithelial tissue respectively that are subject to these mutations. It should be remembered that it is not the fully differentiated cell that undergoes the change into the other kind. The metaplasia is indirect, i.e., there is a preliminary retrogression to a less differentiated type—one earlier in the developmental stage from which the so-called adult cell of the

other kind is developed. This assumption gains considerable weight by the recent observations of Grawitz in the cultivation of tissues *in vitro*, that the intercellular substance in connective tissue resumes the shape of the fixed cell from which it is derived.

Metaplasia takes place as a result of adaptation to altered conditions, as in the case of exstrophy of the bladder, or rider's bone. Chronic irritation and inflammation are the chief factors. It is, therefore, reasonable to suppose, that in an organ so exposed to infection and inflammation as the tonsils, a metaplasia of the fibrous tissue into cartilage and bone would not be a rare occurrence.

INCIDENCE.

With the object of determining the frequency of these findings, a systematic review was made of 750 tonsils in consecutive order, as they were brought to the laboratory from the operating room. Of these, 500 were seen in the pathologic laboratory of the Mercy Hospital, and the other 250 in St. Anthony's Hospital of Denver. Owing to the pressure of other laboratory work, it is the routine practice in these hospitals to section only one of each pair of tonsils for microscopic examination. This has a direct bearing on the percentage of incidences, as will be seen later. After being hardened in formaldehyd solution over night, the tonsils were cut longitudinally and then sections made on the freezing microtome. It may be stated here, parenthetically, that this frozen section method gives excellent results, and enables the pathologist to submit his report promptly to the surgeon. The sections were stained by hematoxylin and eosin, frequently also with Van Gieson's stain. Of the 750 tonsils examined, 54 revealed the presence of cartilage, a percentage of 7.2.

Had the study been extended to the other mate of each pair of tonsils, then, according to the mathematic theory of probability, the percentage would be doubled, and the per cent of incidence would be 14.7. The latter figure approximates the results obtained by Lubarsch, in 1902, from a series of 412 autopsies, in which the tonsils revealed cartilage or bone in 15.77 per cent of the cases. The autopsy material, including as it does a large number of old persons, is not quite identical with the type of cases that come to the hospital for tonsillectomies, among whom children make up a considerable proportion. Nevertheless, the figures approximate closely. They are still below the percentage of 20.9, recently found by Weller in an examination of 1,000

pairs of tonsils. As the latter author rightly concludes, any figures obtained would be below the actual numbers. During the process of sectioning, pieces of cartilage or bone may be torn away and not appear in the stained preparation. Lubarsch, in his series, found 13 per cent of bone occurrence by palpating with the finger tip suspicious looking white strips at the base. This naked eye diagnosis was confirmed by the microscope. This precaution was not observed in our investigation, and some foci of bone and cartilage have undoubtedly been overlooked. It is, of course, out of the question to make serial sections of a sufficiently large number of cases to warrant the time and labor involved. Hence, the figures at best are approximate. They prove, however, that bone and cartilage in the tonsil are of rather frequent occurrence.

No exhaustive statistic study of the age or sex incidence was made in our series. In the series of 250 examinations at St. Anthony's Hospital, there were 14 positives for cartilage, divided equally between male and female. The following table shows the age incidence:

From 5 to 10.....	2
11 to 15.....	1
16 to 20.....	0
20 to 30.....	6
30 to 40.....	3
40 to 50.....	2
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Total	14

The series is too small for drawing deductions, except that, in general, there is seen a greater proportion of positives in adult life. This is borne out in the studies of Lubarsch and of Weller. The increased incidence in later life with accompanying inflammatory changes, tends to support the metaplasia theory.

HISTOPATHOLOGY.

The systematic examination of the tonsils, both gross and microscopically, is becoming more and more a routine practice in modern hospitals. It has encouraged this particular study, and is bound to stimulate further research in the pathology of this organ.

At this scientific gathering, I must yield to the temptation of digressing for a moment from my subject, in order to enter a plea for greater encouragement of these examinations on the part of the laryngologists. A discordant voice is heard now and

then in derision of the utility of microscopic examinations of tonsils. And yet, it is in this very field that more light is needed, and better coordination required from the clinician and the pathologist. In these days, when tonsillectomies are performed largely by the general practitioner and suspicion of unnecessary operating on tonsils is rife, it is essential that histologic norms be established for defining the much abused term "chronic tonsillitis". Routine examinations of excised tonsils will help to bring about a reform.

Returning to the subject, the microscope reveals islands of cartilage in the connective tissue trabeculae of the tonsil. All observers agree that they never arise in the lymphoid tissue proper. Though they may be very near it, their connection with the fibrous tissue is quite evident. There may be one island, but usually there are several, each surrounded by rather dense fibrous connective tissue. In most of the sections, there is an apparent increase of the connective tissue stroma. Evidences of inflammation are occasionally seen in zones of round cell infiltration around the neighboring vessels, or penetrating the adjoining bundles of voluntary muscle fibers or mucous glands. The cartilage cells in some sections are well developed with distinct capsular space. In others, they are more or less compressed, farther apart from one another, and the cartilaginous structure seems to merge by insensible gradations into the peripheral fibrous tissue. One cannot escape the impression that the cartilage has evolved by a process of development from the fibrous connective tissue. In some of the sections, a further progression to bone formation has taken place. There are areas of calcification which stain heavily with hematoxylin. In a few, true bone formation with lamellae and Haversian canals are encountered.

Why the connective tissue in some cases undergoes the metaplastic process into cartilage, while in other cases tissue that is subjected to equal or greater irritation does not, is still beyond our ken. The essence of metaplasia, the internal processes that bring the change about, are unknown. The supposition has been advanced by Nösske, and further elaborated by Weller, that while the theory of aberration of fetal rests is untenable, the connective tissue cells in the region of the second branchial arch may retain at times the developmental or differentiating power to go on to cartilage formation in later life, either spontaneously or as the result of chronic irritation.

That the controversy between the two schools is by no means closed is seen in the last edition of Kaufmann's Pathology, where

a compromise is effected by accepting both embryonal disturbance as well as metaplasia as possible origins of cartilage and bone in the tonsil.

I desire to acknowledge my indebtedness to my associate, Dr. Harry Gauss, as well as to Dr. S. H. Bassow, resident physician at the Mercy Hospital, for their valuable assistance in assembling the data for this study.

BIBLIOGRAPHY.

- Ribbert, H. Lehrbuch d. allg. Pathologie u. pathol. Anatomie, 3rd Ed., p. 465.
 Aschoff, L. Pathol. Anatomie, 5th Ed., Vol. II, p. 723.
 Lubarsch, O. Ueber Knochenbildung in Lymphknoten u. Gaumenmandeln. Virchow's Archiv, Bd. 177, p. 372.
 Ruckert, A. Ueber Knochen und Knorpelbefunde in den Tonsillen. Virchow's Archiv, Bd. 177, p. 387.
 Weller, C. V. The Incidence and Histopathology of Bone and Cartilage in the Tonsil. Annals of Otology, Rhinology and Laryngology, September, 1923.
 Deichert, H. Ueber Knorpel und Knochenbildung an den Tonsillen. Virchow's Archiv, Bd. 141, p. 135.
 Nösske, H. Ueber Knorpel und Knochenbildung in den Tonsillen. Deutsche Zeitschrift für Chirurgie, Bd. 26, p. 559.

DISCUSSION.

DR. W. W. CARTER, New York City: In 1905, I had two cases with the development of bone and cartilage in the tonsil. In one case one tonsil was involved, and in the other both tonsils were involved, and at that time I made a thorough study of the subject and was much interested in it. Since that time, I have not made any microscopic examination of tonsils that were suspected of having bone and cartilage in them, but I feel that the condition is much more common than we have supposed. I have on several occasions taken tonsils in my hand, and by palpation detected hard material which may have been bone. In this connection, I would like to read the following paragraph from my paper in 1905, which was published in the Medical Record, February 4th, of that year:

"Dr. Wright, who examined the specimen, reported as follows: "Microscopic examination of the section made through the entire specimen shows it to be made up largely of fibrous and bony tissue, with a very small amount of lymphoid tissue in it. In the stroma of the organ, a large number of areas of hyalin cartilage are observed, and some of these are plainly being transformed into well organized bone containing Howship's lacunae and lined with osteoblasts and osteoclasts. There are some flat cells associated with the areas of bone formation. In some areas, the bone is being formed directly from fibrous tissue, which at that spot has become as dense as ordinary periosteum; while in others, the transitional stages from fibrous tissue through cartilage to bone can be observed."

Now, gentlemen, the report on the two patients was practically the same. The three tonsils were all sectioned, and microscopic examination showed the same thing. I do not believe this condition is very important from a clinical point of view, but from the point of view of the pathologist it is very important and very interesting. We must distinguish between the deposit of ordinary calcareous substances and the true formation of bone. We must, also, distinguish between the formation of bone and the bone formed from connective tissue around cheesy tuberculous deposits. The process seems to me to be a kind of degeneration. The formation of bone in the chronically inflamed parotid gland and the mammary glands, are instances of where cartilage sometimes forms in connective tissue. In the instance I referred to, the patient was 22 years of age, and had had repeated peritonsillar abscesses for some ten years before the tonsils were removed. Her symptoms were those of chronic tonsillitis. She had difficulty in swallowing, and had repeated peritonsillar abscesses. The tonsil which was involved in the abscesses was the one which contained the cartilage and bone.

DR. WILFRED HAUGHEY, Battle Creek, Michigan: I have had two cases of bone in the tonsil. The first was in 1918, when a doctor in service in France began having quinsy. He had had quinsy for years, first on one side and then the other, and during a lull in activities decided to have his tonsils out. The tonsils were very flat, about once inch in the vertical region and half an inch in the lateral, looked dried up and crusted, with little white spots all over, just like the cheesy deposit in the crypts of the tonsils. This did not wipe off well. On removal, I found true bone in both tonsils, about 0.3 inch thick.

The next case was one I had last spring. This man had also had quinsy, had the same kind of a throat as the former patient, and I found true bone in both tonsils.

DR. L. EMERSON, Orange, New Jersey: We have until recently been in the habit of doing routine tonsil examination all of the time, but there was considerable trouble because of delay in other pathologic reports, and the director of the laboratory said that this was because there was so much work sectioning the tonsils that were sent in, that they could not get the other work out on time. Now the sectioning is done only on request of the operator. The director was glad to do it, but felt that it was unnecessary work, because he knew that unless a man was looking up scientific work it was not being used.

DR. PHILIP HILLKOWITZ, Denver, Colorado (closing): I am thankful to the members who have kindly discussed the paper. I did not expect the Society to get enthused over such an academic affair as the formation of bone in the tonsil, but I did expect to get a rise out of the surgeons as to the necessity of routine microscopic examination of tonsils. Your silence seems to show your agreement with my opinion, which is, indeed, gratifying.

CELLULITIS OF THE ORBIT IN INFANTS AND CHILDREN WITH A REPORT OF TEN CASES.

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For the reason that I disagree with the predominant belief, that orbital cellulitis is more prevalent in adults than it is in infants and children, I am limiting my observations in this paper to the latter. This is exemplified in my own experience in that, during a comparatively short time, I have seen ten of these cases. I am firmly of the opinion that children are very much more subject to orbital cellulitis than has been recognized heretofore. A recent encyclopedia of ophthalmology¹, competently edited, contains the following assertion: "Due to the undeveloped conditions of the sinuses in children, few cases of orbital cellulitis are recorded under nine years of age." With all due respect for this opinion, I believe it should be modified, as nine of the cases I have just referred to were children who had not yet attained nine years of age.

The most frequent and usual cause of orbital cellulitis—I am limiting my remarks to children—is often overlooked, and this by experienced physicians. It is my belief that practically all cases of orbital cellulitis have their origin in disease of the nasal accessory sinuses. Nine of the cases that I am about to detail to you fall within this category. There are several important anatomic conditions of the sinuses and the orbit in children which favor and contribute to the development of orbital complications of sinusitis.

(a) First regarding the bones: There may be congenital dehiscences along the ethmomaxillary suture, in the lamina papyracea, or in the orbital wall of the maxilla¹⁴. The orbital bones are softer in children, ossification not being completed until the sixteenth year. The sutures are ununited until the sixth year.

(b) The orbital periosteum is thin and delicate; it not only lines the openings into the orbit, but forms one of the coverings of the nerves and blood vessels passing through them, thus becoming continuous with the dura mater at the superior orbital fissure¹. This periorbita also extends into the nose through the

lacrimal duct. Schaeffer³ is authority for the statement that at times the lacrimal sac and the mucous membrane of the ethmoid cells are in contact, thus making ethmoid infection and lacrimal sac infection hard to differentiate. In fact, he says there are cases recorded of orbital cellulitis originating in dacryocystitis. The periorbital is easily detachable from the surface of the bone, but it is adherent at the sutures where, in early life, it is continuous with the periosteum covering the opposite sides of the bone.

(c) The sinus mucosa is in most intimate relation with the osseous tissue, so a slight inflammation of one produces a change in the other. Armstrong and Dean⁵ believe, that in cases of equal clinical severity with equally marked changes in the mucous membrane, there is much more apt to be involvement of the underlying bony wall in the child than in the adult.

(d) There is a more profuse development of the lymphatic and vascular systems than in the adult⁴. Infection from the sinuses may enter the orbit along the veins without any bony perforation. Thus the infection in a child's nasal sinus has a much easier path to the orbit than it has in the adult.

The pus does not in all cases perforate into the orbit; it may be guided anteriorly beneath the periorbital until it points through the eyelid, where it sometimes ruptures spontaneously. We are all aware of the frequency of subperiosteal abscesses of the mastoid in children, and should realize that the same condition could occur in the orbit as a result of infection in the nasal sinuses. Valero², of Spain, reports a series of such cases this year.

I do not wish to be understood as stating that an infected nasal sinus is the only cause of orbital cellulitis. There are several other contributing factors, among which may be numbered trauma, carious teeth, osteomyelitis, erysipelas, meningitis, brain abscess, the exanthemata, metastasis or actinomycosis, but these causes are infrequent when compared with nasal sinusitis.

The first symptom of orbital cellulitis to be noted is sudden edema of the eyelids, which in all probability closely follows the actual rupture through the periorbital. The edema may involve the lids of one eye or both. It usually increases so rapidly, that within twenty-four hours the eyelids are so edematous that they can with difficulty be opened. The edema may be accompanied by pain in the eyes, radiating to the forehead.

Chemosis is usually present, and it may be pronounced enough

to cover the entire cornea. Frequently, chemosis may give a valuable clue as to the exact location of the pus in the orbit; if the chemosis is nasal, then the pus will be found in the nasal portion of the orbit; if it is temporal, then look for the pus in the temporal section of the orbit. Following chemosis, proptosis manifests itself, but if the case is seen early enough it is possible to forestall it by proper treatment. But when proptosis is evident, the position of the eye may be a valuable aid in locating the pus. Limitation of extraocular movement is another and frequent symptom, but a history of diplopia associated with it is very difficult to obtain in children. Examination of the fundus usually discloses dilated retinal veins. There may be cases of indentation of the globe simulating detached retina, choked disc, optic atrophy, optic neuritis, retinal hemorrhages, or retrobulbar neuritis^{6 7}, but I have never happened to observe any of them.

The general condition of the child appears serious; the temperature is high, there is a marked prostration, and signs of sepsis are frequently present.

At times, the physician can secure a history of colds or of infectious diseases, but one of the sinuitis is very difficult to obtain from small children. Children seldom complain of nasal discharge, and the diagnosis depends almost entirely upon the examination. As I have pointed out elsewhere^{8 9}, the most valuable single diagnostic aid is the X-ray; but the value of the X-ray in the diagnosis of the orbital condition is limited. Most of my cases disclosed under the X-ray a pansinuitis of one side, and in each case this was confirmed and verified at the time of operation. *Streptococcus hemolyticus* was found in every case in which a culture was made. The importance of an early diagnosis of sinuitis is shown by the fact, that in that stage prevention of orbital complications can often be accomplished, and frequently by nonoperative measures; but once the orbital contents are involved, I believe that the condition should be treated as an emergency and an immediate operation performed. In the case of very small children, intranasal drainage of the orbit is not satisfactory. If free pus has been found in the orbit, it should be drained externally at once, as the orbital tissues disintegrate very rapidly, forming a purulent mass which may lead to total loss of the eye^{10 11}. An external operation should be performed with the following technic:

With the patient anesthetized, the operator sits at the head of the table, which has been lowered to such a degree that the

patient's head rests almost in the physician's lap. Some form of a good head light should be used, and I favor the head mirror. The nasopharynx is packed off. I have found it advisable to place a piece of adrenalin soaked gauze with a thread attached thereto in the middle meatus. This serves as a landmark, and it cannot be forgotten or lost if the string is left hanging out of the nose. A curved incision is made almost identical with that used in the extirpation of the lacrimal sac. Should the sac be encountered, it is lifted from its groove. The periosteum is elevated from the lacrimal bone and the os planum. A perforation



Fig. 1 (Case 1). Age three years. Left maxillary sinusitis and ethmoiditis, frontals not developed. Early orbital cellulitis of left orbit. At operation a perforation was found through the ethmoid bone, with pus in ethmoids and antrum. Complete recovery in two weeks.

is usually discovered through the lacrimal bone or the ethmoid, though the antrum may be the sinus to blame¹³.

A nasal septum retractor is very useful in holding back the eyeball and Tenon's capsule. Pratt's curette is the best instrument I have found to break down the tiny ethmoid cells and to establish a free drainage into the nose. The antrum should be opened from above in very young children, but through the inferior meatus when possible, and the frontal or sphenoid explored when indicated. Drains are left in the nose from the sinuses, and the incision should be left almost entirely closed. Adenoids

should be removed. In twenty-four hours, the drains are removed. In case exophthalmos is marked, considerable care should be taken of the cornea, as repeated dressings of the wound will expose it to possible trauma, which might eventuate in ulcer, with possible loss of the eye.

I have a number of illustrative cases, some of which I will report in detail, whereas others will be given with less particularity. The first is that of a boy, three years old, referred to me by Dr. Max Seham (Fig. 1). His lower eyelids were red and swollen following a cold. There was no chemosis or exophthalmos present, but there was considerable nasal obstruction, associated with a purulent discharge; the temperature was 100° and the general condition of the child seemed good. Dr. Seham prescribed the use of the nasal suction pump, which gave relief, and he seemed to be doing well. After about ten days, the right upper eyelid suddenly became edematous. The edema rapidly increased to such an extent, that it was absolutely impossible for him to open his eye. There was a copious discharge from the right nostril. Temperature was $102\frac{1}{2}^{\circ}$. I was called in and found that his middle meatus contained pus. Transillumination disclosed that the right antrum was dark. An X-ray examination of the sinuses made by Dr. Allison revealed a clouding of the right ethmoids and antrum, with absent frontal sinuses. The eye was quiet, no chemosis, proptosis or limitation of movement; but the patient complained of pain when he rotated his eye. The fundus was negative. A diagnosis of acute sinusitis, with early cellulitis of the orbit, was made and an immediate operation was performed, following the technic suggested above. A perforation was found through the ethmoid bone, but no free pus was found in the orbit. The ethmoid cells and antrum were filled with thick pus. These cavities were curetted and drained through the nose, the wound being completely closed. In twenty-four hours the temperature was normal, and in two weeks a complete recovery resulted.

My second case is that of a ten year old girl, who complained of pain in the right eye, accompanied by swelling of the right upper eyelid. (Fig. 2.) The pain started about twenty-four hours before her admission to the hospital, followed by swelling twelve hours later. No history could be secured of any contagious disease or of any other illness preceding the present trouble. There was no history of nasal discharge, and the child did not experience headaches. At the time of admission, the temperature was 103° , the pulse was 116, and respiration was 16. The child had a

toxic appearance, and both right eyelids were very edematous and red, although the left lids were but slightly puffy. Chemosis was very marked in the right eye, and proptosis was so advanced that there was very little power of rotation in any direction; but no diplopia was complained of. Vision was 20/30 in the right eye and 20/20 in the left. The right cornea was clear and the pupil reacted readily. The fundus revealed dilated veins, but no other pathology was in evidence. Examination of the nose revealed no pus, perhaps because it was draining into the orbit,¹⁵ but in an X-ray examination, all of the right sinuses were found to be cloudy. A diagnosis was made of orbital cellulitis, but it was



Fig. 2 (Case 2). Ten-year old girl. Right pansinusitis with orbital abscess. Operation disclosed pus in the orbit, a perforation through the orbital plate of the ethmoid, pus in ethmoids, frontal and antrum. Recovery in three months, during which time two metastatic abscesses were treated.

hoped that a stab incision through the upper eyelid would produce sufficient drainage to give relief. This was found to be insufficient, in that it drained the orbit with no effect upon the sinuses. The next day a radical external operation was deemed necessary, performed, like my first case, after the technic referred to above. Thick pus was found, under pressure, in the frontal, all the ethmoids and the antrum. Drainage of these cells, and of the orbit, also, was brought about through the nose, the external wound being closed.

It was necessary to treat the child for three months before she recovered, during the course of which a metastatic abscess developed in the right ankle, as well as one in the scalp, both of which required incision and drainage. The eye, itself, was injured by reason of the dressings coming into contact with the cornea and producing an ulcer, which, in spite of treatment, progressed to perforation, adherent leucoma resulting. The sinuses cleared up quite completely.

The next case is that of an infant, a year and a half old. (Fig. 3.) A week before admission to the hospital, swelling was noticed



Fig. 3 (Case 3). 1½-year old infant. Left pansinusitis with definite clouding of the orbit. Operation showed orbital abscess, pus in ethmoids, antrum and sphenoid. Recovery in thirty days, during which time a metastatic abscess developed in the right ankle.

in the left cheek, which traveled to the outer half of the upper eyelid. The eyelid rapidly increased in size, becoming very red and inflamed. The family physician, upon being consulted, incised the eyelid, but located no pus. The infant was then taken to the hospital. His temperature was 98.6°. The left eye was pushed forward, downward, and outward. The upper bulbar conjunctiva was chemotic; the cornea was ulcerated; the W.B.C. was 42,000. Examination of the nose disclosed no pus and no external excori-

ation. Upon the sinuses being X-rayed, a left pansinusitis and a cloudy orbit were found. Diagnosis of orbital abscess and sinusitis was arrived at, and a radical external operation was performed which confirmed the diagnosis, as the antrum, ethmoids and sphenoid all contained thick pus. We were unable to check the ulcer of the cornea, which ultimately perforated, resulting in loss of the eye. During the course of the thirty day treatment that followed, a metastatic abscess developed in the right ankle, which required incision and drainage.

My next four cases are so similar in all respects, that they can properly be grouped together. One patient was five months



Fig. 4 (Case 4). Age six years. Right ethmoids and antrum cloudy. Frontals not fully developed. Orbit slightly cloudy. Operation disclosed free pus in orbit, with perforation through ethmoid. Antrum drained from above. Recovery complete.

old; two were six years of age, (Fig. 4.) the other was seven. (Fig. 5.) In each case, there was sudden swelling of the lids, chemosis, exophthalmos and temperature. X-ray of the sinuses disclosed that the ethmoids, antrum and orbit of the affected side were all involved; and this was true of each of these four cases. In each case, the typical operation was performed and a perforation found into the orbit from the ethmoid cells, with free pus in the orbit. All recovered completely without visual defects. My next case resembles these four, with the exception, that during the course of the operation, cerebrospinal fluid escaped through



Fig. 5 (Case 5). Age seven years. Orbital cellulitis with maxillary and ethmoid infection. Three operations required before results were obtained.

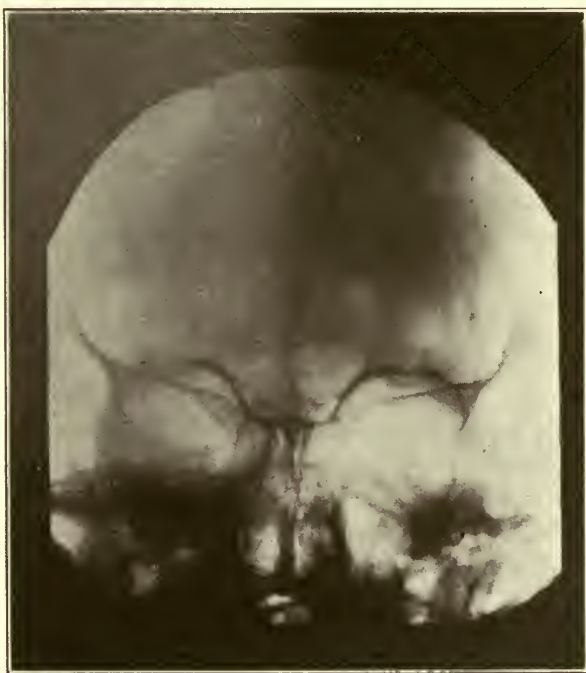


Fig. 6 (Case 8). Age nine years. Left orbital abscess, left pansinusitis. Operation confirmed diagnosis. Cribriform plate perforated at operation with escape of cerebrospinal fluid. Recovery after one month.

the cribriform plate, although no deleterious results eventuated.

Case nine is that of a two year old girl, referred by Drs. Huenekens and Moriarity. She was in her second week of scarlet fever, when the left eyelids suddenly became red and swollen. She had considerable headache and much purulent nasal discharge from the left nostril. Her temperature was 101°. Examination revealed that the tonsils were well removed; much pus was seen running down the postpharyngeal wall. The left preauricular gland was enlarged, also the left cervical glands. Much pus could be obtained by suction of the left nostril. The eye showed no chemosis, proptosis or limitation of motion; vision normal; pupil regular and reacted; fundus showed slight engorgement of the retinal veins. A diagnosis of acute sinusitis was made, and the suction pump prescribed. Great improvement followed the use of suction, and the lid edema disappeared in forty-eight hours. Four days later the symptoms returned, with chemosis starting at the caruncle, and bulbar conjunctiva slightly edematous. Operation was advised but refused. Hot compresses were used, and in the morning the eye was full of pus and the swelling had gone down. Under the upper lid, near the inner canthus, the point of rupture was found. For a month the child progressed well, when a sudden recurrence of all the symptoms appeared. Chemosis was more marked, temperature 102°, and the child seemed much worse generally. Operation was performed as outlined above. A perforation through the ethmoid bone was found, but no pus was in the orbital cavity. This was a subperiosteal abscess which had produced orbital symptoms due to serous exudation and vascular changes, but no actual infection of the orbital contents. The antrum was opened through the inferior meatus and drained into the nose. A large mass of adenoids was removed. The child had very little reaction to the operation and progressed to an uneventful recovery.

The last case that I will present, is that of a male child, three years of age, referred by Drs. Schlutz and Stewart. (Fig. 7). His first symptom was severe headache, not preceded by a cold or any other illness. The right temporal region was somewhat swollen, but no earache or discharge were noted. At the end of two weeks, this swelling disappeared, followed by exophthalmos of the right eye, accompanied by marked chemosis and edema of both eyelids. The temperature was high, but there was neither convulsions nor paralysis. The exophthalmos increased until, at the end of the fourth week, a fistula appeared at the external canthus, discharging pus, which gave considerable relief. During the next

two weeks, Jacksonian convulsions took place, beginning in the left arm and becoming generalized. During the next month, but little change was noted; the child still complained of severe headache, exophthalmos persisted and the fistula continued to discharge. The patient was brought to Minneapolis to Drs. Schultz and Stewart, who found a rigidity of the neck, with a positive Koenig sign, a positive Brudzinski sign, and sluggish knee kicks on the right and normal on the left. The spinal fluid was found to be under but little pressure, clear, contained 12 cells, positive Nonne and a negative Wasserman. The W.B.C. was 15,000 with 61% P.M.N.'s; the urine was negative; the temperature was 99°.



Fig. 7 (Case 10). Age three years. Sinuses clear on X-ray but orbit cloudy. Orbital cellulitis from a brain abscess which drained through the orbit, opening externally at the outer canthus.

At that time I was called in. I found the right upper eyelid edematous, but not sufficiently so to prevent the eye from being opened. Exophthalmos was present, the eye apparently being pushed directly forward. Upon palpation of the globe, no resistance was encountered in the orbit. The pupil was slightly dilated and reacted readily to light; the cornea was clear. Extraocular movements were limited laterally, but were good in other directions. The fundus disclosed dilated veins, without choked disc. A small fistula at the external canthus was discharging pus, through which opening a probe was readily passed without either

increasing or decreasing the volume of pus emitted. The nasal sinuses, in the X-ray, were found to be clear, although the orbit was cloudy. The nose, throat and ear examinations were negative. It was deemed advisable to explore the orbit, which was done under a general anesthetic. Necrotic bone was found at the apex of the orbit, but no abscess cavity was encountered. The orbital roof was intact. The patient reacted very slightly to this exploration, and there was no appreciable change in his condition. Early in the morning of the second postoperative day, the child was awakened by a severe headache, which was not susceptible of control by morphin. He was entirely rational, rolled over in bed, and died. A postmortem was performed by Dr. Charnley McKinley, of the University of Minnesota. A large temporal lobe abscess was found, which had perforated the orbit at the superior orbital fissure, remaining external to the periorbita, extending the entire length of the orbit and breaking through at the external canthus. A localized meningitis surrounded the abscess; the middle ear and mastoid contained seropurulent material, and the tympanic membrane was thickened, but intact.

It is known that a brain abscess can break through the orbit and produce an orbital abscess, but I have been unable to find a report of any such case in the literature. Dandy, in a personal communication, writes that he has never seen such a condition. As the sixth nerve passes through the superior orbital fissure, one can readily see how it could become involved in this case; and the continuity of the periorbita with the meninges also explains how such an accident could occur without the orbital contents being involved.

In conclusion, orbital cellulitis is more frequent in children than has been recognized heretofore; and this is accounted for by reason of the anatomy and development of the nasal sinuses and the orbit. The most frequent cause of orbital cellulitis is infection of the nasal sinuses. If this is recognized early enough, the orbital complications can often be prevented. Radical treatment should be resorted to as early as possible after the diagnosis of orbital involvement is made, thus curing the sinuses, conserving the vision, and, in many cases, saving the life of the patient.

BIBLIOGRAPHY.

1. Am. Encyclopedia of Ophthalmology.
2. Valero. Arch. de Oftal. Hispano-Am., May, 1923, v. 23, p. 261.
3. Schaeffer. The Nose and Olfactory Organ. Blakiston, 1920.
4. Oppenheimer. Jour. A. M. A., vol. 73, No. 9.
5. Dean. University of Iowa. Studies in Medicine.

6. McCullough. Trans. N. Y. Acad. of Med., Oct. 26, 1921.
7. White. Quoted by Dean.
8. Phelps, K. A. Sinuitis in Children. N. Y. Med. Jour., Sept. 22, 1917.
9. Phelps, K. A. Sinuitis in Children. Journal-Lancet, Nov. 1, 1920.
10. Skillern. Accessory Sinuses of the Nose.
11. Hajek. Ztsch. für Hals, Nase and Orheilk., Berlin, April 25, 1923, v. 4, p. 511.
12. Scammon. Prof. of Anatomy, U. of Minnesota. (Personal Communication.)
13. Dean. Annals of Otol., Laryng., and Rhin., 1923, p. 285.
14. Whitnall. Anatomy of the Orbit.
15. Vail. Trans. Am. L. R. and O. Soc., 1919.
16. Scammon. Prof. of Anatomy, U. of Minnesota. (Unpublished Research.)

DISCUSSION.

DR. L. W. DEAN, Iowa City, Iowa: I want first to take the opportunity of congratulating the speaker upon his paper. He has not only had a wealth of material, but he has worked it up splendidly, and has presented it in a way which is instructive, at least to me. I am very pleased because his paper illustrates the incidence of paranasal sinus infection in infants and young children in the upper Mississippi Valley, which seems to be a favorite spot for this disease. There is a goiter belt, and there seems to be a sinus belt, and Dr. Phelps and I seem to be in the sinus belt for infants and young children. I have never seen an infection in the frontal sinus in a child under eight years of age, and I would be inclined to exclude, not definitely but roughly, the frontal sinus as a cause of infection in infants and children under eight years of age. Dr. Phelps showed one case ten years of age, where there was involvement of the frontal sinus. The sphenoid sinus is very precocious. I think the greatest advantage to be secured from an X-ray picture of the sinuses in infants and children with ocular complications, is to determine the size of the paranasal sinuses. The sphenoidal sinus, as I said, is very precocious. Dr. J. Parsons Schaeffer has a specimen in his collection showing a sphenoidal sinus at birth. At the age of five years and five months, I have seen, at autopsy, a diseased sphenoidal sinus 18 mm. wide.

In orbital cellulitis, in children, secondary to paranasal sinus disease, we have clinically two types of cases—first, those where the sinus infection is pointing at the inner angle of the orbit or in the cheek, and second, those cases where there is no pointing. I operate the paranasal sinuses by intranasal procedures. The ophthalmologist then waits twenty-four or forty-eight hours, and drains the orbit or leaves it alone as seems best. Frequently the intranasal work on the paranasal sinuses is all that is necessary. Where there is pointing of an abscess externally, the same intranasal procedures are used, and in addition simple external drainage is established.

I believe that in osteomyelitis secondary to paranasal sinus disease in infants and young children, where there is an involvement of the external table of the face, we must adopt two surgical procedures. First, be sure there is no collection of pus which is not drained. We must have drainage, and some of these cases require two, three or even four operations for drainage. If I had a patient with osteomyelitis

of the maxilla secondary to sinus infection, I would drain externally where it was pointing, and I would watch that patient, and if I suspected that it was not draining sufficiently, I would drain again. We have had cases where it has been necessary to drain three or four times during six months. I think Dr. Phelps mentioned a case where he found it necessary to do three operations. Second, I do not think we should injure the bone in this class of cases. If there is a sequestrum present, it should be removed, but the limiting barrier which Nature has formed should not be disturbed.

DR. WILLIAM C. POSEY, Philadelphia, Pa.: Some years ago, I called attention to a class of cases in which orbital cellulitis in children was occasioned by disease of the superior maxillary bone. In some of these, the bone disease was secondary to a general blood infection; in others, it resulted from an infection of the alveolar border, the inflammation being apparently conveyed to the alveoli by an infected nipple during the act of suckling. The alveolar border once involved, suppurative periostitis soon extended rapidly under the periosteum along the anterior surface of the upper jaw, or through the maxillary antrum to the orbit. As a rule, an acute osteomyelitis of the entire superior maxillary follows, with all the signs of an active inflammatory process, such as is observed from whatsoever cause. The inflammation in the orbit may extend along the periosteum posteriorly, be most intense, and invading the sphenoidal fissure, may implicate all the nerves of the orbit, both motor and sensory.

Not infrequently the optic nerve becomes implicated, and the ophthalmoscope may reveal the signs of neuritis, and if the inflammation be continued, of atrophy. After a variable period of time, but generally rather rapidly, cure may follow by absorption, and the affection may disappear without leaving a trace. More usually, however, spontaneous evacuation follows the suppurative process, and fistulae persist in the orbit and in the mouth. In consequence of the former, cicatrices affect the lid and ectropion results. At times the entire lower rim of the orbit may slough away, leaving a most disfiguring, indrawn scar. As is usually the rule in osteomyelitis, the sequestrum shows but little tendency to separate itself from the living bone for a long time, so that healing is frequently very protracted.

In other cases, alveolar disease may be present in children and fistulae form, without there being the slightest swelling of the cheek. This is attributable to the peculiar anatomic structure of the alveolar process in early life, in consequence of which the pus from the alveoli gains ready access to the orbital cavity, exciting there a purulent inflammation of its contents, without the body of the bone being involved in a general osteomyelitic process. Investigations have proven that the antrum is quite large enough to become the seat of inflammation even in the earliest months of life. In an admirable dissertation, Guisez has given the clinical picture of maxillary sinusitis in the infant, and attributes the common or acute form to grippal coryza, to involvement of the mucous membrane of the sinus during convalescence from infectious disease, or, in fine, to any form of nasal infection. Comparatively recently, Beauvois has summed up the entire subject in a masterful manner, and suggests that in very young infants a leucorrhoea of the mother may readily infect

the nose of the infant, or the contagion may be conveyed through the medium of the bath or even of the air.

Regarding the frequency of orbital periostitis from all causes in children, Birch-Hirschfeld maintains that it is comparatively rare, for of 298 cases of orbital periostitis which he had had under his observation, but 33 (11.1 per cent) occurred during the first decade of life, the affection being met with in the second and third decade twice as frequently. Vossius, on the other hand, maintains that it shows a special predilection for children. My own experience tallies with that of Birsch-Hirschfeld, for I have met with the condition but rarely.

Treatment consists in early evacuation of the pus from the orbit, and in establishing proper drainage from that cavity and through the body of the superior maxilla if it, too, be the seat of disease. If the abscess point beneath the mucous membrane, a bistoury should be inserted into the fornix at its most dependent point, and the pus allowed to flow away; but if the abscess is diffuse, the incision should be made through the skin, due regard being given to the area of greatest resistance, which should be determined by careful palpation of the orbit.

DR. F. PARK LEWIS, Buffalo, N. Y.: While orbital cellulitis is met with occasionally in the practice of every ophthalmologist, I am sure that Dr. Phelps has had a much richer experience in the number of cases that he has seen than occurs with most of us. It is not always necessary that this condition be attacked through the nasal passages. In a case which has recently come under our observation, in which there was marked proptosis, a fixed and deviated eye, tense lids and congestion of the retinal veins, one of our ablest rhinologists reported freedom from any nasal or sinus involvement. The condition had followed an attack of influenza. In that case, we made a deep incision into the orbit through the upper lid, with no relief. Following that, another deeper incision was made through the lower lid, to a depth of an inch and a half, and this was followed by an evacuation of pus, a fistula forming which persisted for two months. Then, a second operation was done, in which necrotic bone was removed. Following that, the fistula healed and the patient recovered.

It evidently was a pus infection arising from ethmoidal sinusitis. The sinus had cleared, but had meantime developed an orbital cellulitis, which was relieved by drainage.

DR. JAMES MOORES BALL, St. Louis, Mo.: I wish to call attention to one etiologic factor not mentioned, and that is trauma at delivery. In Modern Ophthalmology, you will see a picture of an infant, aged ten days, who was injured during birth, and whose right orbit was filled with pus. Obstetric injuries of the eye have not been given the attention I think they should receive in our American literature. Our friends in England have done much more toward reporting eye injuries at birth than we have. These obstetric injuries may range all the way from a slight, transient edema of the cornea, to an actual destruction of the eyeball. The late Mr. Simeon Snell, for instance, had brought to his clinic an infant, one day old, whose eye was hanging on its cheek as the result of trauma received during instrumental delivery.

DR. V. A. CHAPMAN, Milwaukee, Wis.: I wish to say just one

word of warning regarding diagnosis in these cases. I have seen one case very recently which was erroneously diagnosed as an acute dacryocystitis, and in those cases which point at the inner canthus or on the lower lid, such a thing may happen.

DR. W. H. ROBERTS, Pasadena, Calif.: I, too, wish to say just one word in regard to diagnosis, and that is, that in all cases of exophthalmos presenting in infants and children, in whom a positive indication for pus in the orbit cannot be seen, a differential examination of the blood should be made. A malignant disease which often arises in the accessory sinuses and is known as chloroma, in its beginning will present nearly all of the signs of an infection such as this paper deals with. If a differential count is made, it will show at once whether it is chloroma, and will prevent our doing an operation which could only result disastrously, for chloroma is absolutely incurable. In the American Journal of Ophthalmology, this month, I have reported a case of chloroma which occurred in my practice last year, in which this diagnosis could very readily have been made during its course, but the blood examination revealed the true condition.

DR. JOSEPH C. BECK, Chicago, Ill.: There is one point in the causation of sinus disease in infants which has not been mentioned, but which is very important, and that is traumatism produced by incompetent men in general practice. Following an exanthematous disease like measles, scarlet fever and so on, if the child develops symptoms related to the orbit, headaches and so forth, these gentlemen often proceed to sound the upper straits of the infant's nose, and as a result there occurs a typical picture of orbital cellulitis, although there is no evidence of any pus in the nose at all. Another point, in one such case, although the indications were quite clear for doing something radical, a pediatrician was called in when the child was already developing meningeal symptoms, and in view of this fact, the indications for operating upon the orbit were disregarded, and the child was not operated upon and died.

Another point I wish to make, is regarding radical operation and immediate closure, as reported in the paper. I think too much importance was placed upon the immediate closure. Although I believe in clearing up all the other sinuses that may be infected, I think a cigarette drain should be inserted through the outer angle of the frontal sinus during the first forty-eight hours, or used through the inner angle of the orbit, so that drainage may not be blocked.

DR. KENNETH A. PHELPS, Minneapolis, Minn. (closing): Many important points brought out in the discussion are covered in the paper, but through lack of time I could not read it in full.

I agree with practically everything Dr. Dean said, and the idea he brings out regarding intranasal operation, I think applies particularly when the orbital contents are not involved, as in a subperiosteal infection. I think the prophylactic side of this thing is very important. If we would recognize that sinusitis does exist in young children, and would look for it more frequently, we could prevent many of these complications.

There was one thing I did not read regarding the cause of this trouble, and that is actinomycosis. Dacryocystitis in young children can easily be mixed up with an ethmoiditis, and Schaeffer says that an

ethmoiditis may be caused by a dacryocystitis.

As to Dr. Beck's point about drainage, I put twenty-four hours as the time at which the drain should be removed.

I wish to emphasize again the prophylactic side. If we recognize the sinus infection early in these children, we often can treat it without operation and prevent these complications altogether.

CONTRIBUTING FACTORS IN THE ETIOLOGY OF MYOPIA. A PRELIMINARY STUDY.*

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OMAHA, NEBRASKA.

While there are many different views as to the cause of myopia, the mechanical principles underlying the elongation of the globe would seem clearly to point to a loss of strength, resiliency or hardness of the sclera.

The globe, not being supported anteriorly and posteriorly by the tonicity of the ocular muscles, gives way in these directions, especially at the posterior pole. What the underlying conditions are which cause this weakening of the sclera, it may be impossible to prove experimentally, as the progress of myopia extends over too many years. Clinical observation, therefore, must be given due weight in the understanding of this problem.

As the antrum, the sphenoids, ethmoids and frontals are all in close relationship to the orbit, we would expect disease of these sinuses to be frequently associated with disorders of the orbital contents, and it is the clinical relationship of disease of the sinuses and myopia to which I wish to call attention.

The recognition of the etiologic factors in head conditions will depend largely on the acknowledgement that all forms of chronic catarrh and atrophic rhinitis are essentially nasal sinus infections in various grades of activity. I have referred to this in previous studies¹⁻²⁻³⁻⁴. Though this belief is gaining ground with rhinologists, it is still far from being universally accepted. For four or five years, the view that all chronic nasal infection comes from the sinus infection has seemed to me the only possible explanation for the different manifestations of chronic catarrh of the nose and nasopharynx, and since adopting this axiom, not only has the treatment of these conditions been very much more satisfactory, but the explanation of associated phenomena has been clearer.

The diagnosis of sinus disease does not essentially mean that operation is necessary for relief: many cases do better without operation, proper attention being given to general and nasal hygiene.

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It has been routine with the author for the past three years in every refraction case to examine personally also for teeth, tonsil and nasal infections. The findings drew his attention to the almost invariable association of nasal infections and acquired myopia. While only a small proportion of nasal infection cases are myopic, the reverse is not true. Almost every case of myopia was found to have latent or manifest nasal infection or catarrh, or a history of some severe nasal infection. The nasal infection is considered invariably due to nasal sinus disorders of either latent low grade infections or more manifest processes.

It is possible to determine that sinus disease exists from:

(1) *History.* All exanthemata and severe upper respiratory infections, severe colds in head, grippe, influenza, involve not only the nasal cavities proper, but also the nasal sinuses. These infections may go on to complete resolution with return to health, or if the infection has been unusually prolonged or severe, a chronic, low grade, latent infected condition of the sinuses will remain.

(2) *Symptoms.* In the absence of tonsils and adenoids, mucus or mucopus chronically in nose or nasopharynx, or atrophic, dry nose, or recurring redness of pharynx, indicate a reservoir of infection in the sinuses. Enlarged adenoids are a symptom of nasal and nasal sinus infection.

(3) *Neighborhood Symptoms.* Ear inflammations, purulent or catarrhal, indicate an existant or previous sinus infection (if infected tonsils and adenoids have been excluded). Chronic conjunctivitis, recurrent attacks of bronchitis, asthma and pneumonia are strongly suggestive of residual upper respiratory infection.

(4) *X-ray.* Opacity of sinuses is corroboratory evidence, but many times a transparent nasal secretion may be highly infectious and toxic, and the case not show any shadow.

In the following histories, the age quoted is that of the patient when first seen.

CASE REPORTS.

CASE 1. HELEN C. 8 Years old.

2-11-14. Complains of vague pain in eyes and headaches. Vision: R. 20/20, L. 20/20. Homatropin: R. $+ .25 \times 75 = 20/20$; L. $+ .25 \times 105 = 20/20$. Fundus normal. Media clear.

2-9-15. Had scarlet fever. Released from quarantine two weeks ago. Has pain above eyes and headaches. Sees floating gold balls and black specks. Vision: R. 20/20, L. 20/20. Fundus showed fine spots of degeneration at the macula region. Mucopus in the nose. Spray prescribed.

8-14-16. Had chicken pox two months ago. Vision: R. 20/30, L. 20/30. Homatropin: R. $-25\text{C}-50\times 120=20/20$; L. $-25\text{C}-50\times 60=20/20$.

4-28-20. Vision: R. 20/200, L. 20/200. Homatropin: R. $-2.00\text{C}-.75\times 105=20/20$; L. $-2.50\text{C}-.50\times 75=20/20$.

Tonsils infected. Mucopus in nose and throat. Treatment refused.

Patient with hyperopic astigmatism and infection of nose, left by scarlet fever, develops in six years myopia of over 2 D.

CASE 2. W. R. W.

First seen 1906 when 33 years old, 3-12-06. Myopia has increased since that time from R. $-1.25\text{C}-1.75\times 90$, L. $-1.25\text{C}-1.25\times 90$ to R. $-2.00\text{C}-2.25\times 90$, L. $-1.25\text{C}-2.25\times 85$, (12-10-19).

Patient has always had nasopharyngeal catarrh.

6-14-19. X-ray showed both antra cloudy, though patient had no acute exacerbation of his chronic nasal condition at this time.

In this patient a chronic nasal antrum infection is associated with increasing myopia.

CASE 3. F. C. A. Age 53.

11-7-21. Patient comes for change of glasses. Wearing -5.00 O. U. Accepts R. $-5.75=20/40$, L. $-6.00=20/50$. Floating opacities in vitreous. Nose shows scabs in middle fossae. Pharynx congested. Patient says he has always had a "dry catarrh" and has worn glasses since early childhood. X-ray shows right antrum cloudy $++$. Left antrum $+++$. Other sinuses clear.

This patient has had long standing myopia associated with long standing, low grade sinus infection.

CASE 4. Mrs. P. C. D. Age 30.

4-23-23. Complains of pain behind eyes and over bridge of nose. Comes for change of glasses. Homatropin: R. $-2.00\text{C}-.75\times 165=20/20$; L. $-1.50\text{C}-.50\times 15=20/20$. Dust opacities on post. lens surface. Fundus normal. Tonsils and teeth out. There are green scabs in nose and pus in pharynx, and there is a stale odor on breath. Patient says she has suffered from catarrh for years. X-ray: antra and ethmoids slightly cloudy, frontals clear, but poorly developed.

Patient with a chronic low grade sinusitis, other head focal infections (teeth and tonsils having been removed), and also with a low grade uveitis (dust opacities), has associated with these a low grade myopia.

CASE 5. Helen M. Age 8.

9-22-17. Comes for change in glasses. Wearing R. -1.25 , L. $-.50$. Fitted two years ago. Accepted under homatropin: R. $-2.00 \text{ C} - .12 \times 180 = 20/20$; L. $-1.00 \text{ C} - .50 \times 15 = 20/20$. Each year the myopia has increased and glasses have been regularly changed. The patient is surrounded by healthy environment and uses eyes moderately and under good conditions. The last examination, in 1922, showed: R. $-4.50 \text{ C} - .62 \times 165 = 20/15$; L. $-2.50 \text{ C} - 1.50 \times 15 = 20/15$. In 1918, examination of the nose showed patulous nasal cavities with scant mucopurulent secretion. Tonsils small and infected.

Patient with mild grade of atrophic nasal catarrh and diseased tonsils shows progressive myopia in spite of good general hygienic care of the eyes.

CASE 6. Mrs. M. C. Anisometropia.

Patient, 32, (when first seen 13 years ago) has worn since then with only slight changes: R. $-2.50 \text{ C} - .75 \times 170$, L. $+.50 \text{ C} + .25 \times 15$. Has always had colds in the head since childhood, and always much more severe on the right side. In 1913, I removed the ant. portion of the right middle turbinate to obtain better drainage for an infected right ethmoid. In 1914, I operated on the right mastoid for acute mastoiditis. In 1923, persistent discharge from the nose continued for over two months. X-ray showed both antra $++$. Drainage openings were made in the inferior meatus, and condition cleared up under treatment. The right antrum had much more marked infection than the left.

Patient with anisometropia, with history of head infection for several years, the more marked infection being on the myopic side.

CASE 7. Barbara H. Age 8.

3-24-19. Low grade uveitis. Spots on Descemet's membrane. Nasopharynx congested and red with mucopus. Vision: R. 20/200, L. 20/100. Local and constitutional treatment.

4-5-20. Descemet's almost clear. X-ray shows left antrum slightly cloudy. Vision: R. 20/20, L. 20/20. Homatropin: R. $-.25 \text{ C} - .25 \times 135 = 20/15$; L. $-.25 \times 45 = 20/15$.

12-26-22. Has continued local treatment. Descemet's clear. No increase in refractive error. Nasopharynx, condition greatly improved.

Young patient with symptoms of nasal sinus infection and uveitis, on improvement of nasal and ocular condition shows slight myopia, which did not increase.

CASE 8. Allen S. Age 14.

5-1-23. School nurse discovered that he did not see well with his right eye. Vision: R. 20/200, L. 20/20. Homatropin: R. $-1.50=20/20$; L. Unimproved, 20/20. Fundus and media clear. Tonsils have been removed. Teeth are clear. Nose is blocked and nostrils thickened. Nasopharynx has swollen lymphatic pillar on right covered with mucopus, draining from right nose.

Boy with evidence of right sinus infection has right myopia.

CASE 9. Harold R. Age 16.

12-31-20. Fundus and media clear. Vision: R. 20/30, L. 20/30. Homatropin: R. $-.50\bigcirc-.25\times 105=20/20$; L. $-.50\bigcirc-.25\times 165=20/20$.

Nose obstructed by deflected septum. Pharynx congested. Tonsils are out. Teeth clear. X-ray: Antra and ethmoids are slightly cloudy.

7-7-21. Operation. Submucous and ethmoid operation, with opening into both antra. Local treatment.

8-9-22. Refraction of eyes: R. $-.50\bigcirc-.75\times 180=20/20+$; L. $-.50\bigcirc-.37\times 150=20/20+$.

Boy with low grade myopia had evidence of sinus trouble. On correction of nasal condition, myopia only slightly increases.

CASE 10. Robert D. Age 18.

4-8-16. R. $-2.00\bigcirc-.75\times 90=20/30$; L. $-.50=20/20$. Has always been subject to colds.

2-6-23. Nose: Turbinates swollen. Pharynx swollen and red. Tonsils: Pus expressed. X-ray shows ethmoids cloudy.

4-17-23. Has repeatedly refused operation. Has been under nasal treatment at intervals for several years. Homatropin examination shows no increase of myopia since 1916.

The myopia of old age, at least partly due to swelling of the lens, may also be included in this etiology.

CASE 11. O. C. H. Age when first seen, 56 years.

6-19-11. R. $+.50\bigcirc+.37\times 180=20/15$, L. $+.25\bigcirc+.75\times 90=20/15$.

Eight years later, patient became myopic in both eyes. There are vitreous opacities, and opacities on the posterior surface of the lens. Teeth show apical infection. Tonsils have inclusions of pus. Nose has mucopurulent crusts. Tonsils and dental disease removed. Local treatment for the nose.

12-27-22. Myopia has increased to: R. $-4.00\bigcirc-1.25\times 90=20/30$; L. $-2.25\bigcirc-1.50\times 165=20/30$. Vitreous remains cloudy. Patient cannot be prevailed upon to give attention to his dry catarrh.

No. Patient	Age first seen	Date	Refractive Error			Date last seen	Refractive Error			Nasal Condition.
			Sph.	Cyl.	Axis		Sph.	Cyl.	Axis	
1. Man	31	3-11-23	— .25	— .75 — .37	75 75					Puffiness under eyes. Mucopus discharge in nasopharynx.
2. Man	42	5-19-05	—2.00 —2.25	— .25 — .25	110 75	3-16-23	—3.75 —4.00	— .62 —1.25	110 75	Mucopurulent secretion in nose.
3. Woman .	72	5-18-23	— .75 — .75	—1.00 —1.00	180 180					Frequent colds. Mucopus in nose. X-ray: Rt. Ant. ++. Lt. Ant. + Ethmoids and Sphenoids +
4. Woman .	35	5-21-23	— .50 — .75	+ .87 +1.25	80 105					Postnasal discharge. Marked catarrhal deafness.
5. Woman .	40	5-30-23	—2. —2.	— .25 — .50	180 180					Turbinates atrophic. Transparent, sticky, musty smelling secretion.
6. Woman .	65	2-10-10	+ .25 — .50	+ .50 — .25	180 135	4- 2-23	+ .50 — .75	—1.25	180	Swelling over Lt. antrum. Mucopurulent secretion in both nasal cavities.
7. Woman .	69	12-19-07	Emmetropic Emmetropic			6-13-23	— .50	— .62 —1.00	180	Mucopurulent discharge from nose and pharynx.
8. Woman .	23	4-10-23	— .25	— .25 — .50	90 60	Fitted elsewhere previously R. + .50 L. + .50				Trans. secretion in both middle fossae. Has history of previous acute antrum trouble. X-ray: Lt. Ant. ++
9. Boy Case 8. Previous Report.	14	5- 1-23	—1.50	Emmetropic						Turbinates swollen and nostrils thick. Pharynx swollen. Red streak on right.
10. Man	46	12-30-15	— .75 — .25	— .25 — .50	110 70	5- 4-23	—1.25	— .75	140	Mucopus in nose and postpharynx.
11. Woman ..	64	5-11-23	—2.00 +2.25	+ .25	30					Mucopus in nose. X-ray: Antra ++
12.*Girl	14	7-28-23	— .50	— .37	180	5-15-20	—3.00 —3.75	—1.00 — .62	90 90	Atrophic turbinates. Mucopus in nose.
Girl	14	9-14-21	—4.50 —4.50	— .25 — .25	15 150					Nose seems clear.
Girl	15	5-27-18	—1.50 —2.00	— .50 — .50	60 75					Pus in nose and nasopharynx. X-ray: Rt. Ant. ++. Lt. Ant. +
13. Woman . Case 6. Previous Report.	35	2- 2-10	—3.00 + .25	—1.00 + .50	155 180	5-16-23	—2.75 + .50	— .62 + .25	160 15	Transparent secretion from nose. X-ray Antra ++. Other sinuses clear.
14. Man	67	12-15-20	+1.25 +1.25	—5.00 —4.00	90 80	5-17-23	+1.25 +1.25	—4.50 —4.00	95 75	Mucopus in nose and pharynx.
15. Boy	10	5-24-23	—3.25 —2.75							Nose full of mucopurulent secretion. Two months previously I operated upon him for acute mastoiditis on right side.
16. Man	25	6- 2-23	— .25	— .25 — .50	60 90					Nose seems clear.

*While only the first girl comes in this sequence, the other two are her sisters, and the findings are interesting, as the sisters also showed nasal infection.

Old patient with evidence of chronic, low grade sinus infection shows change from hyperopia to myopia.

The above cases have been quoted in skeleton to give as briefly as possible the main findings. The preceding table shows cases taken consecutively in my personal practice.

De Schweinitz⁵ says he has published some observations which indicate that as the result of choroiditis, myopia may rapidly develop in certain cases, observations which are in accord with the statements of Kneis, Priestly Smith, and others, and that occasionally after an acute illness, especially one of the exanthemata, a myopia develops, or there may be a sudden increase in a pre-existing myopia.

Edridge-Green⁶ thinks the primary and essential cause of myopia is an obstruction of the outflow of this fluid (posterior lymph), which causes myopia by elongation of the eyeball, and that it is evident that any cause tending to diminish the resistance of the sclerotic will be an important factor. He thinks, also, that heredity may be a special weakness of the sclerotic to give way under pressure, and he gives as exciting causes, measles, whooping cough, bronchitis, and advises prevention of congestion of venous circulation of the head.

Blegvad⁷ says heredity and near work explain prevalence of myopia, and says correction has no influence on the progressive course of myopia.

Huguenin⁸ states that a congenital abnormal lack of resistance of the posterior segment causes the myopic changes, but also states that the inquiry among 150 cases of anisometropia gave results which were not exactly favorable to the law of inheritance, and thinks we could change little in the favorable course of a developmental period of the individual.

Risley⁹ thinks myopia occurs through the turnstile of astigmatism, and that astigmatism produces a distension of the sclera consequent upon the hyperemia of the uveal tract brought about by increased eyestrain.

Koster¹⁰ says the myopic eye has as much lens power as the hypermetropic or emmetropic eye, and only if full correction prevents the patient from blinking, and thus pressing his eyes by the lids, can it be regarded as a remedy.

Arthur Wood¹¹ thinks the difference in the ciliary muscle of the myope and the hypermetrope the cause, rather than the result of myopia, and as this variation is inherited, the whole of myopia is hereditary.

Fuchs¹² states, that as elongation of the eyeball is a normal process in its development, myopia develops either because the elongating force is excessive, or because the resistance offered is too weak, and says in all probability both factors are concerned, but the deficient resistance of the eye would seem the more important of the two.

Duane¹³ quotes Stilling, Hasner and Weis, stating that the external muscles in convergence cause pressure, venous congestion and elongation of the globe.

These opinions may be analysed, and the relation between the different factors and nasal sinus infection shown.

The sclerochoroiditis and resulting weakened sclera (de Schweinitz, Koster, Edridge-Green, Kneis, Priestly Smith, Fuchs) frequently follow severe infective conditions of the nose and its cavities. The exanthemata (especially measles) and severe colds are the most prolific cause of both active and latent sinus disease in children. They all involve the upper respiratory tract, and as the acute infection subsides, there frequently remains a low grade, latent infection in the sinuses, of which the antrum in children is the most developed. The very nature of this residual infection could be expected to produce the changes characteristic of myopia, a chronic, low grade choroiditis with weakening of the sclera.

The near work and eyestrain of Risley, Harman and Duane, carrying with it sedentary life and unhygienic conditions, is detrimental to the relief of sinus disease, and by the congestion of the eye, such strain increases the bad effect of the neighboring infection.

The heredity of Hugenin, Blegvad, Edridge-Green, Wood, may be influenced by the frequency with which the mother may transmit nasal infection to her children. The frequency with which the mother may transmit common colds or acute nasal infections has been recently noted by Winholt and Jordan¹⁴, but I have often noted that a mother with chronic nasal infection has children also suffering from nasal infection.

The obstruction to lymphatic drainage (Edridge-Green) and to the venous circulation at the posterior pole of the eye probably often accompanies a neighborhood congestion of the sinuses, which may be considered the source of the congestion. Tortuosity of the retinal veins is often seen accompanying nasal infections.

That the first step in the sinus infection may be often nutritional is suggested very strongly by the recent investigations of Daniel¹⁵ et al., in which nasal sinusitis was produced constantly

in animals by a diet deficient in fat soluble vitamine A. These investigations point the way to dietetic, prophylactic and progressive treatment of both sinuitis and myopia. The diet of all children should be well supplied with food containing the fat soluble vitamine A.

If this view of the etiology of myopia is proven, there will be much that can be done to prevent its progress. The obligation to correct carefully refractive and muscular errors, and care of proper light, position, etc., will remain as important as ever. If, however, during the developmental period, proper treatment is given to the nose, the child can be carried through this period and go on to normal development. I have given my views previously showing how such treatment can be carried out, and that operative work on sinuses is generally not indicated³⁻⁴.

In conclusion, therefore, all the factors which have been considered in the production of myopia may be dependent on the devitalizing effect of a neighboring nasal sinus infection, active or latent, and either acquired from the mother, or the result of the exanthemata or any other severe infection of the upper respirator tract, and probably having as an underlying factor the lack of vitamine A in the diet.

This preliminary study is presented with the hope that suggestions may be received from other observers, which may enable us to discover the truth regarding the part nasal infections play in the production of myopia.

REFERENCES.

1. Lemere, H. B. The Antrum as the Main Source of Nasal Catarrh. *The Nebraska State Medical Journal*, Nov., 1920, vol. V, No. 11, p. 326.
2. Lemere, H. B. A New Operative Technic for the Operative and Postoperative Treatment of Maxillary Sinus Diseases. *The Larynoscope*, St. Louis, May, 1922.
3. Lemere, H. B. The Diagnosis of Nasal Sinus Disease in Children. *The Archives of Pediatrics*, September, 1922.
4. Lemere, H. B. The Treatment of Accessory Nasal Sinus Disease in Children. *American Journal of Diseases of Children*, vol. 24, pp. 520 to 525.
5. De Schweinitz, George. *Diseases of the Eye*, 8th edition, 1916, p. 129.
6. Edridge-Green, F. W. *The Lancet*, January 26th, 1918, vol. 1, p. 137; *The Lancet*, March 15th, 1921, vol. 1, p. 469.
7. Blegvad, O. *Ugeskrift for Laeger*, Copenhagen, February 14th, 1918, vol. 80, p. 287.
8. Sidler-Huguenin. *Archives of Ophthalmology*, Nov., 1916, vol. XLV, p. 577.
9. Risley, Samuel D. *Ohio State Medical Journal*, Aug., 1919, vol. 15, p. 467.
10. Koster. *Zeitschrift f. Aug.*, vol. 34, p. 215; *The American Encyclopedia of Ophthalmology*, vol. XI, p. 8266.
11. Wood, Arthur. *Ophthalmoscope*, July, 1916. *The American Encyclopedia of Ophthalmology*, vol. XI, p. 8270.

12. Fuchs. Textbook of Ophthalmology, 7th edition, 1923, p. 196.
13. Duane-Fuchs. Textbook of Ophthalmology, 7th edition, p. 197.
14. Jordan, E. O., and Winholt, W. F. Journ. A. M. A., v. 81, No. 4, p. 280.
15. Daniels, Amy L., Armstrong, Margaret E., and Hutton, Mary K. Journal American Medical Association, Sept. 8th, 1923, vol. 81, No. 10, p. 828.

DISCUSSION

DR. JAMES M. PATTON, Omaha, Nebraska: I must confess to a feeling of some misgiving when Dr. Lemere told me of his opinion that the sinus was an etiologic factor in the causation of myopia. This was somewhat excusable, because we have a great deal of sinus infection in our section of the middle west, and I wondered whether he might not be confusing coincidence with etiology. I was further excused, perhaps, because on looking over a number of titles regarding the etiology of myopia, I saw frequent allusion to exanthematous diseases, tuberculosis, endocrin disturbances, etc.—in other words, general diseases which react more or less directly on development. The time was too short to consider any large number of clinical cases, so I took one hundred cases of myopia of one diopter or more, just as they came in the files, and one hundred similar cases of hyperopia of about the same degree, and examined them for history of sinus involvement. Unlike Dr. Lemere, it has not been our custom to examine the sinuses in all cases of refraction, but we do examine all cases in which the history or symptoms seem to point to anything of this kind, and I was surprised to find that in the one hundred cases of myopia, sixty-six gave evidence of some sinus condition of sufficient importance to call for careful examination. The remaining thirty-four cases may or may not have had some latent or previous very mild sinus infection such as Dr. Lemere has referred to.

I then turned to my hyperopes, and was surprised to find that the ratio was reversed. Of the hyperopes, only twenty-five cases gave evidence of any sinus involvement, while seventy-five were free so far as history was concerned. They, of course, were subject to the same reservations as the thirty-four myopes, but the ratio is suggestive, to say the least.

Dr. Lemere, from the title of his paper, gives us to understand that this is a preliminary report, and he does not claim that all cases of myopia are due to sinus disease, and certainly the one hundred cases from our records do not prove anything, but it seems to me that Dr. Lemere has brought a very important suggestion to us, and if, as he says, these conditions do influence the progress of myopia, then it throws on us the responsibility of having our myopic cases very carefully examined for sinus trouble and the conditions corrected.

DR. WILLIAM HARDIN SEARS, Huntingdon, Pa.: Dr. Lemere is to be complimented in presenting, for the consideration of this Section, a possible contributory factor in the etiology of myopia, with a series of cases to illustrate and substantiate his findings.

That axial myopia is due to elongation of the globe, especially its posterior half, was mentioned as early as the Seventeenth century and at various times thereafter, until Arlt¹ gave his masterly discussion of

the problem in 1856. Since then, this relationship has been generally recognized and accepted.

Many theories to account for this elongation have been brought forward, all admirably buttressed by statistics—consanguinity, near work, accommodation, convergence, configuration of the orbit, abnormal insertion of the ocular muscles, unusual shortness of the optic nerve, eye-strain, astigmatism, inflammatory disease of the globe, constitutional disease, heredity, psychic element, age, sex and race.

The earlier theories were largely extrinsic in character, although Parsons quotes Mauthner's statement: "The congenital basis of myopia is to be sought in nothing else than in a defective resistance of the sclerotic, especially in its posterior half." It is now almost three decades since R. D. Batten gave out his theory of the development of myopia through constitutional diseases, which acted by inducing nutritional defects in the sclera. In 1901, Priestly Smith² advanced much the same thought, in that constitutional disease might promote yielding of the sclera, and assuredly did aggravate morbid choroidal and retinal change. Ochi³, in 1919, suggested that the sclera be considered part of the cranium. In this way, children with a weakened constitution, especially the scrofulous, may suffer from a less resistant sclera, as a direct consequence of a general skeletal weakening or disease. Sonder⁴, in 1921, assigns the acute diseases of childhood as factors in the evolution of progressive myopia. He states that frequently myopes ascribe the beginning of their ocular trouble to some acute infectious disease, a fact not generally accepted by the ophthalmologist. He presents statistics bearing out this theory. So many observers have reported cases of myopia appearing after acute infectious diseases, as to lend plausibility to this thought.

Inflammatory disease of the ocular tissues, especially chorioretinitis, as a part of the myopic process was brought forward by von Graefe.⁵ He later, however, modified his opinion. Shon⁶ says choroiditis always accompanies scleritis, and Parsons⁷ states the converse may well be true. At all events, in recent years a number of students have reported the development of myopia in the presence of chorioretinal disease.

For a number of years, the close association of ocular inflammation with nasal disease, including of course the paranasal cells, has been discussed in the literature. The earliest suggestion of nasal disease influencing myopia, I have found, was that of R. D. Batten, who wrote of this connection early in the last decade. There may have been others whom the writer has overlooked. Turner⁸, in 1918, in discussing such relationship, reports a case of progressive myopia checked only by the treatment of the ethmoid cells. Edward Jackson⁹, in 1919, in discussing the etiology of myopia, mentions among other points to be investigated that of nasal disease. It would seem, therefore, that for a number of years there has been some trend of thought toward the association of constitutional disease with the development of myopia, and of comparatively recent date, a similar association with nasal disease.

The genesis of myopia cannot be viewed from any one angle, for one or several of the factors accepted as influencing its development may be present without its occurrence; on the other hand, it may develop where not one of these causes can be found.

Its study then must be of sufficient breadth to cover all the various

factors which have been generally accepted as contributing in any way to its development.

Steiger¹⁰, in his striking article, reminds us that the problem is a biologic one; that emmetropia, hypermetropia and astigmatism are just as much a part of a process of heredity as is myopia; that the problem must be considered phylogenetically, not ontogenetically.

Straub's¹¹ hypothesis of a psychic element entering into the production and maintenance of emmetropia and hyperopia as well as myopia, is both ingenious and interesting.

The essayist's conclusion, that some of the factors which have been considered in the production of myopia are probably dependent on the devitalizing effect of a neighborhood nasal sinus infection, active or latent, and either acquired from the mother or the result of the exanthemata or any other severe infection of the upper respiratory tract, can hardly be considered definitely proven; but that nasal sinus disease may have a direct bearing on the evolution of myopia, will in the light of the present study, be generally admitted.

How great a share inflammation of the paranasal cells has in the genesis of myopia will be known only after an exhaustive study of this, as well as of all the other factors generally accepted as causal in its production, and when a critical analysis and comparison of these findings has been made, then, possibly, the varying shade of importance may be assigned to each contributing factor.

In analyzing a case or a series of cases, it may be well to bear in mind: That at birth a very small percentage of infants are myopic; at school age this percentage has increased to possibly double; from this time to puberty, or about the age of sixteen, during the development period, the increase is steady; during this period more cases are developing, and of those with myopia already present, many are increasing the amount; at puberty, or the close of the rapidly developmental epoch, myopic progress may cease; at any time, progressive myopia may stop its onward course and remain stationary for a varying period; the amount of myopia may decrease; myopic increase occurs largely during the developmental age, and myopic decrease is much more frequent later in life, after the age of thirty for instance.

Interesting statistics which have direct relation to these facts are those of Jackson and Clarke. Edward Jackson¹², in 1902, presented a study of one hundred and twenty-three myopic eyes, which had been under his personal observation for periods varying from three to seventeen years. These were refracted at intervals during this time and a full correction worn. It is probable that few, if any, reports covering all the points of similar value included in this survey, are to be found in the literature.

The myopia remained stationary in 75.6 per cent, diminished in 5.7 per cent, and increased in 18.7 per cent. In but six eyes was visual acuity diminished. E. Clarke³ reviews seven hundred and fifty cases of myopia, excluding high myopia with gross fundus change, observed during the forty years of ophthalmic practice. Of these, 50.6 per cent remained stationary, 13.4 per cent decreased. 1.1 per cent of those decreasing were over thirty years of age; 18.7 per cent increased not over .50D; 17.3 per

cent increased .75D and over; but 2.1 per cent increased over 2.00D, and but .07 per cent increased 4.00D.

Owing to the limited time given for the presentation of Dr. Lemere's paper, it was obviously impossible for him to cover many of the details in his case reports, which, as he states, are quoted in skeleton. This makes a review more difficult to one discussing the paper and less fair to the essayist, in that doubtless many of the points brought up have already been considered by him, and omitted by reason of lack of space and time.

CASE 1. A girl with slight hyperopic astigmatism developed, between the ages of eight and fourteen years, a myopia of R.—2.00D. and L.—2.50D., each combined with a low grade astigmatism. She had scarlet fever at nine years and whooping cough at ten. Degenerative spots in the fundus were noted immediately after scarlet fever. Important points not mentioned are those of heredity and the general physical condition of the child. Likewise, it must not be forgotten that this myopia arose during the developmental age. Another question of equal importance in this discussion, is whether the acute and chronic infections may not frequently affect the optic tunics directly, as they probably do at times, instead of indirectly through contiguous nasal sinus disease. In this case, fundus changes were noted immediately following scarlet fever.

CASE 5. A girl between the ages of eight and thirteen years developed an increase of myopia of R. — 2.50D. combined with c. — .50D., and L.—1.50D., combined with c.—1.00D. No mention of heredity, previous medical history or the general physical condition of the child is made. In view of atrophic rhinitis at this age, the question of constitutional dyscrasia might well enter in. The increase of myopia, in this case also, occurred during the developmental age.

CASE 8. Another girl, eight years of age, with uveitis and very low myopia. The etiology of the uveitis is not suggested. The refractive error could very well be due to changes in the aqueous associated with the Descemetitis, although this in turn could be the result of sinus disease.

CASE 3. Man of fifty-three years, with long standing myopia associated with long standing sinus infection. This case is cited without detail, except to show a myopia in childhood with a history of associated nasal disease. No mention is made of organic disease associated with vitreous opacities to account for his decreased vision. This brief reference to the case reports is made to call attention to a very few of the points which must be considered in any case, and not to invalidate Dr. Lemere's findings, for as noted above, he has very likely already covered these and others.

The extremely short time available for preparation of this discussion rendered anything but the most superficial approach out of the question. The same reason prevented the report of some cases, bearing on this question to the extent of an associated nasal or tonsillar disease in the presence of myopia.

If the essayist's paper arouses the members of the section to an increased interest in the study of nasal disease in its relation to ocular inflammation, and especially to any association it may have in the genesis of myopia, it will prove a valuable production indeed.

To close—In the etiologic study of a given case of myopia, one may well remember the last paragraph of Jackson's¹⁴ editorial on the etiology of myopia, which is quoted verbatim. If a school girl develops myopia, has she astigmatism? Is she converging excessively? Is there a sclero-choroiditis present? Has she vascular or nasal disease? Is there evidence of defective nutrition in other directions, particularly of skeletal or other connective tissue structures? When we can answer all these questions, we may be able to assign to each factor its proper share in the result. Meanwhile, we must devote attention to the remedy of all those conditions that seem likely to be important factors in producing elongation of the globe.

BIBLIOGRAPHY.

1. Arlt. *Krankheiten des Auges*, III, 1856.
2. Priestly Smith. *British Medical Journal*, Oct. 19, 1901.
3. Ochi, S. *Am. Jour. Oph.*, vol. 2, p. 675.
4. Sonder. *Arch. d'Ophth.*, vol. 37, p. 290.
5. von Graefe. J. H. Parsons, *Pathology of the Eye*, vol. 3, p. 926.
6. Shon. *Ibid.*
7. Parsons. *Ibid.*
8. Turner, H. H. *Penn. Med. Jour.*, vol. 22, p. 427.
9. Jackson, E. *Amer. Jour. Oph.*, vol. 2, p. 700.
10. Steiger, A. *Zeit. f. Augenh.*, vol. 36, p. 127.
11. Straub. *Oph. Year Book*, 1909, p. 297.
12. Jackson, E. *Ophthalmic Record*, 1902.
13. Clarke, E. *Proc. Royal Soc. Med., Sec. Oph.*, 1921.
14. Jackson, E. *Amer. Jour. Oph.*, vol. 2, p. 700.

TRANSACTIONS
OF THE
TWENTY-EIGHTH ANNUAL MEETING
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Academy of Ophthalmology
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OPHTHALMOLOGIC DIVISION

JENSEN'S RETINITIS.

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In 1917, the essayist reported a typical case of Jensen's retinitis, with a review of all available literature up to that time. In a careful survey of the literature from 1917 to May, 1923, the author has been able to find but one new case of "Jensen's Disease", and that was reported by K. Kusama, in the *Zeitschrift fuer Militaer-Aertze* (published in Tokyo, Japan), in 1918, no. 75, 1. This report was not available, therefore no further reference will be made to it in this paper. The author's second case came under observation, on March 24, 1921. The patient was a tall, slender, overgrown, country boy of 18, who complained of blindness in the left field. Ophthalmoscopic examination revealed a typical retinitis of the Jensen type, the single circumscribed lesion being situated very near the papillary margin, in the upper nasal quadrant. At that time the inflammation was limited to the retina and had been manifest to the patient for about one week; there was no evidence of uveitis; the refractive media were perfectly clear; the external appearance of the eye was normal; vision 20/30, with a sharply contracted field, showing a sector defect as described by Jensen and others. A careful biologic examination, together with urinalysis, was negative in every respect, the report is appended herewith:

Wassermann—Negative; R.B.C.—4,850,000; Hb—75%; Malaria—Negative; Sputum—Negative; von Pirquet—Negative.

Family history negative with the exception of a younger brother, who had lost an eye at 5 years, through primary detachment of the retina followed by a general uveitis, which necessitated enucleation. The patient stated that he had always been well, but was just recovering from an attack of influenza when he discovered the eye disorder. No focal infection could be demonstrated in the teeth, tonsils or accessory sinuses. There was no pain, lachrimation or photophobia, and as treatment has proven of little or no benefit in these cases, and the patient lived at some distance in the country, the only treatment instituted was one drop of one per cent solution of atropin in the left eye, once a day with a drop shade. The right eye was unaffected. One week later, there was an active uveitis in the affected eye, with cloudy

vitreous and some exudates on the lower quadrant of Descemet's membrane; vision C.F. at 10 ft. This continued until the fundus was entirely obscured by vitreous opacities and an exudate covering the entire circle of Descemet's membrane, reaching its peak about April 24th, four weeks after the first visit to my office and probably six weeks from the beginning of his disorder.

Resolution was slow but steadily progressive. On May 10th, the fundus was dimly visible, Descemet's membrane had perceptibly cleared, and the single focus of retinochoroiditis could again

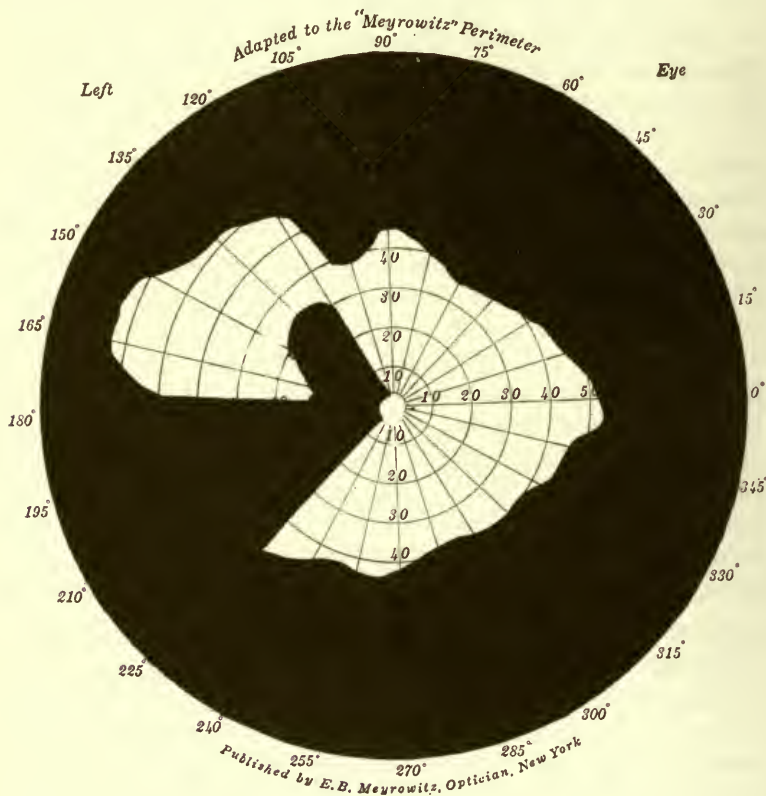


Fig. 1. Jensen's retinitis. (1st week.)

be outlined. I saw the patient at intervals until January 1922; by that time he had made an excellent recovery. The lesion had resolved itself into a choroidal atrophy, the sector defect remained unchanged. There was marked contraction of the field as a whole, with a single scotoma corresponding to the site of the lesion. Vision was 20/30. This case differed from my former one in that the uveitis became general, with dense vitreous exudates and deposits on Descemet's membrane. It differs also in

that the apex of the sector defect does not correspond with the site of the lesion. This has not been observed in any of the former reports.

Since the case report of 1917, the author has taken the field in every case of circumscribed chorioretinitis coming under observation, without finding the sector defect in any case other than the two cases reported as Jensen's disease. Recently, a young woman came in for refraction, who had a single circumscribed

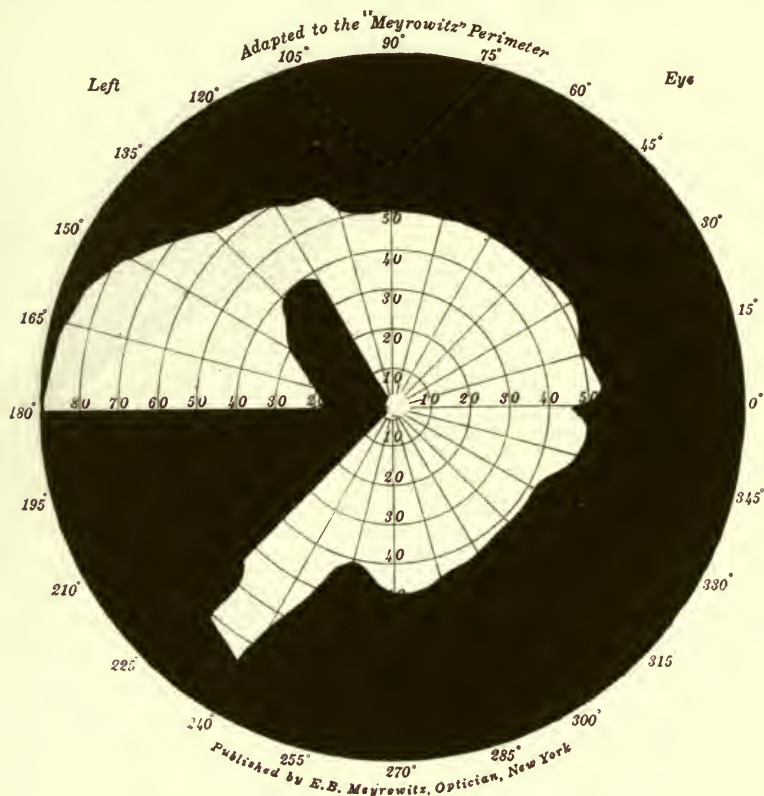


Fig. 2. Jensen's retinitis. (10 months later.)

choroidal atrophy of long standing situated near the papilla on the temporal side, presenting the typical appearance of old choroiditis centralis, with apparently a complete atrophy of the temporal half of the disc. It occurred to me that if this case did not show a sector defect in the field, the theory advanced by Rönne, "That all cases of acute choroiditis are examples of Jensen's disease" would have to be abandoned, and that Jensen's disease must be recognized as a distinct pathologic

entity, the exact pathology of which has not yet been demonstrated. This case, as will be shown on the lantern slides, did not show the sector defect which distinguishes Jensen's Retinitis from other types of circumscribed retinochoroiditis. An attempt has been made also by means of lantern slides to reproduce the lesion as it appears to the observer in Jensen's Retinitis as compared with a simple choroiditis centralis. Also, a comparison of the field charts is shown.

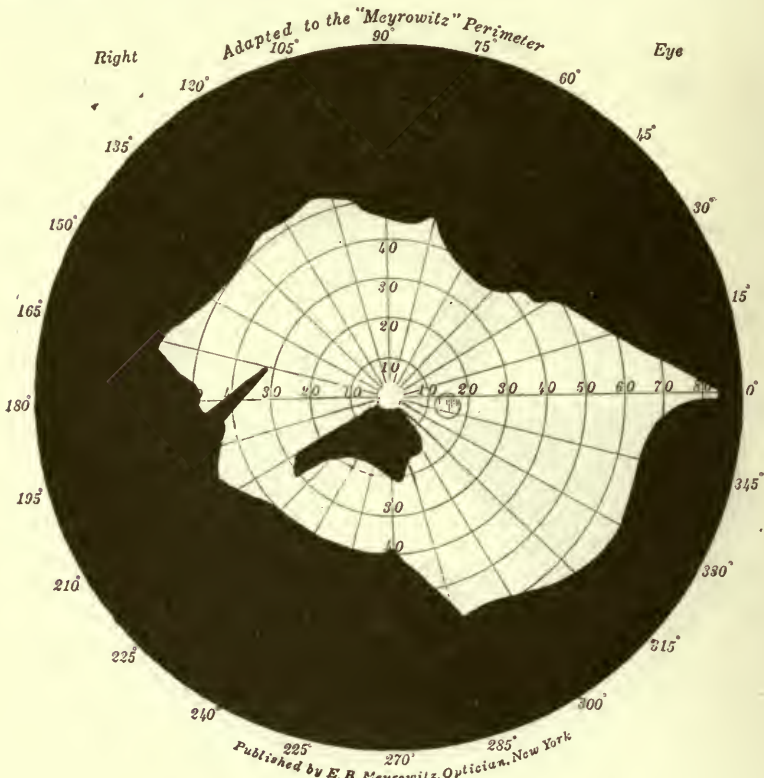


Fig. 3. Choroiditis centralis. (Old.)

In the author's opinion the conclusion seems inevitable, that if Jensen's Retinitis is a pathologic entity, it is comparatively rare, not more than one in each one-thousand of miscellaneous eye diseases showing the characteristic sector defect in the field.

It would seem also that treatment is of little avail, either in shortening the course of the disease or in modifying the end results. However, certain complications, such as the macular hemorrhage in my first case, seem to yield to intensive elimination.

BIBLIOGRAPHY.

- Appleman, L. F. *Am. J. Ophth.*, St. Louis, 1914, v. XXXI, pp. 60-62; also *Ophth. Rec.*, Chicago, 1914, v. XXIII, pp. 221-223.
- Blessig, E. Ein Fall von Retino-Choroiditis juxtapapillaris (Edmund Jensen) S. Chorioretinitis parapapillaris. *Arch. f. Ophth.*, Leipz., 1910, v. LXXIV, pp. 284-289.
- Jensen, Edmund. Retinochoroiditis juxtapapillaris. *Arch. f. Ophth.*, Leipz., 1908-1909, v. LXIX, pp. 41-48, 2 pl.
- Groes, Peterson. Retinochoroiditis (Edmund Jensen). *Klin. Monatsbl. f. Augenheilk.*, Stuttg., 1912, n.F., v. XIV, pp. 159-186.
- Ham, A. Ein Fall von Retinochoroiditis juxtapapillaris (Edmund Jensen). *Klin. Monatsbl. f. Augenheilk.*, Stuttg., 1914, n. F., v. XVIII, pp. 487-493.
- Van der Hoeve, J. Nervenfaserdefekte bei Retinochoroiditis juxtapapillaris (Edmund Jensen). *Klin. Monatsbl. f. Augenheilk.*, Stuttg., 1914, n. F., v. XVI, pp. 487-493.
- Ormond, A. W. Case of Retinochoroiditis Juxtapapillaris. *Proc. Roy. Soc.*, London, 1912-1913, v. VI, *Ophthalmol. Sect.*, pp. 16-19.
- Patterson, J. A. Retinochoroiditis Juxtapapillaris. *Ophth. Rec.*, Chicago, 1910, v. XIX, p. 260.
- Rönne, H. Ueber die Retinochoroiditis (Edmund Jensen). *Klin. Monatsbl. f. Augenheilk.*, Stuttg., 1915, v. LIV, pp. 455-461.
- Verhoeff, F. H. Ein Fall von Syphilom des Optikus and der Papille mit Spirochaetenbefund. *Klin. Monatsbl. f. Augenheilk.*, Stuttg., 1910, v. XVIII, p. 315.
- Verhoeff. A Case of Syphilitic Retinochoroiditis Juxtapapillaris, with Microscopic Examination. *Tr. Am. Ophth. Soc.*, Phila., 1916, v. XIV, pp. 568-577, 1 pl.; also *Arch. Ophth.*, N. Y., 1916, v. XLV, pp. 352-358, 1 pl.
- Martin, H. H. Retinochoroiditis Juxtapapillaris (Jensen's Retinitis). *Journal Southern Medical Association*, June, 1918, pp. 455-459.
- Kusama, K. Ein Fall von Retinochoroiditis Juxtapapillaris (Edmund Jensen). *Zeitschrift fuer Militaer-Aerzte*, Tokyo, 1918, No. 75, p. 1.

DISCUSSION.

DR. EDWARD B. HECKEL, Pittsburgh, Pa.: Jensen's retinitis, or Jensen's disease, also described under the more formidable, but perhaps more exact, title of retinochoroiditis juxtapapillaris, is certainly a rare condition, or else has gone unnoticed by many observers. The distinguishing physical characteristics of this condition are that there is a distinct lesion of the fundus at or near the optic disc, and that the field of vision shows a sector like relative scotoma extending well out to the periphery of the field, with its apex corresponding to the lesion of the retina, at or near the normal blind spot. Whether central vision is involved or not depends upon the direction and extent of the retinal lesion; if it should happen on the temporal side of the disc, near the horizontal meridian, we certainly would have good reason to expect involvement of central vision.

The ophthalmoscope shows a circumscribed white swelling at the disc, the retinal vessels more or less obscured, not at all unlike opaque nerve fibers, with the rest of the fundus negative or hazy.

The sector like defect in the field of vision is strongly suspicious of vascular changes in a branch or branches of the arteria centralis. Whether these vascular changes are primary or secondary is difficult to answer. This condition at least renders the sector of the retina

supplied by the small branch or branches involved in the retinal lesion functionless.

It would seem that the cause of this condition might reasonably be an infection of some sort; lues or tuberculosis has not yet been demonstrated as a causative factor. Focal infection may play an important role.

Typical cases might go unnoticed for some time, as they would cause little or no symptoms. Some of the cases reported show a fluid vitreous with floating opacities, some circumcorneal injection, a well pronounced Descemetitis, or endothelial deposits on the cornea, or, in other words, a general uveitis which is so atypical, that it is a question whether it should be classified under this title at all. It would seem that the only cases which should properly come under this classification are those that show the definite fundus picture described, with the rest of the eye quiet and negative, and the corresponding sector like defect in the field of vision.

We owe our thanks to Dr. Martin for again calling our attention to this rare and most interesting condition, and may we hope that it will stimulate further and more exact observations, so that the near future may establish this as a definite and distinct condition, with a characteristic syndrome entirely its own?

DR. GEORGE E. DE SCHWEINITZ, Philadelphia, Pa.: As the essayist has said, Jensen's retinochoroiditis is comparatively rare, although perhaps not quite so rare as statistics would indicate. My personal experience with this disease comprises three patients, two girls, one 8 years old and the other not over 12, and a man in the early thirties.

Pathologic investigations, for instance, those of Verhoeff, indicate that the disease is primarily of retinal origin, the choroid being secondarily involved. He and Igersheimer suggest it may be sometimes of luetic origin, but this is almost certainly not frequently the case. Tuberculosis has also been advocated as an etiologic factor. One of my patients improved rapidly after the elimination of focal infections in the teeth and tonsils, and another, a young girl, after the relief of a chlorotic anemia, but whether these foci of infection and this anemia were really causative I am not prepared to say. In all of the patients under personal observation, the lesions were confined to one eye, and I have not seen the type described by Groes-Petersen, associated with pigment changes in the opposite eye, or that variety reported by Van der Hoeve, with macular lesions in the corresponding eye. In all these instances, the defect was interpreted by a sector like fault in the visual field, extending from the blind spot to the periphery (diagram exhibited). The photograph of the fundus herewith exhibited is by Dimmer, and shows the lesions very well.

DR. EDMOND E. BLAAUW, Buffalo, N. Y.: I want to add to the recent literature a very important article in the Bericht ueber die 43ste Versammlung der deutschen ophth. Ges., 1922, by Fleischer (Erlangen), who suggests multiple sclerosis as a possible cause. Wagenmann stated that his impression is that in most cases the origin is in the choroid, and only secondary in the retina.

EARLY DIAGNOSTIC RETINAL SIGNS OF TUBERCULOSIS.

FREDERICK F. TEAL, M.D.

LINCOLN, NEBR.

AND

ERLE B. WOODWARD, M.D.

MONROVIA, CALIF.

In advancing the ideas put forth under this title, we have met with considerable opposition on the part of our colleagues, some of whom insist that there are no signs of beginning tuberculosis in the retina that are characteristic. It is a common opinion among physicians and oculists, that in certain localities it is a waste of time to look for tuberculosis, particularly for evidence of it in the eye. Having for the past six years made a study of this subject, both in a locality that is supposed to have very little, Lincoln, Nebraska, and in one in which there is an abundance of cases, Monrovia, California, we are prepared to state unequivocally, that in a certain type of individuals, there is very little difference in the frequency of these manifestations when it comes to locality. You will find many cases in both localities. Stark, in a paper read at New Orleans in 1920, made a plea for a greater effort among oculists to be on the lookout for tuberculous disease in the retina, and our excuse for writing this paper is to emphasize his stand in this regard.

We think you will accept without question, that a large percentage of all persons have been at one time or another affected with tuberculosis. Numerous authorities could be cited to show this to be a fact. Would it be unusual, therefore, to find evidence of it with comparative frequency in our examination of the retina? On the contrary, it would be strange if we did not. How then can you explain the fact that the most intelligent and highly trained group of the medical profession, the ophthalmologists, have not seen early evidences of tuberculosis in the eye more often in this large class of individuals? Our contention is that they have seen them time and again, but have not realized their significance. This has been our individual experience and no doubt that of others. We are of the belief: That certain phenomena as seen with the ophthalmoscope, which have been classed as "normal," are not normal. That certain ocular changes

taken together are evidence of beginning pathologic changes that are significant and point to tuberculosis. That these signs appear so frequently that it is possible to make a diagnosis of tuberculosis before the discovery of tuberculous lesions elsewhere.

Notable contributions to the subject of tuberculosis have been made by Stock, Axenfeld, Fleisher, Fuchs, Zentmayer, Jackson, Finnoff, Stark, Lewis, von Hippel, Bogardus, Tooke, Herter, Buck, Natalie, Patterson and many others. They report numerous changes due to tuberculosis; lowered vision, blindness, exudates of varying degree in the retina, changes in the blood vessels, tubercle, vitreous changes, hemorrhages, retinal detachments, swelling of the disc, iris involvement, exudates in the cornea and aqueous, choroidal changes, etc.

In our investigations of tuberculous patients, we have found certain signs that appear to be fairly constant; in examining hundreds of school children who were under par, some of whom afterward developed tuberculous lesions, we found these same signs. We found them in children of parents who had died of tuberculosis. Briefly the signs are these:

Areas in the retina looking like shot silk.*

A pale straw colored area along the vessels starting from the disc. It looks like a thinning out of the normal pink color of the retina immediately contiguous to the vessel wall, and gives the appearance of a sheath around the vessels.

Fracture lines in the vitreous. These show as shining threads, appearing usually parallel with the large vessels. They appear and disappear when you move your mirror from side to side, as in using the retinoscope. They come and go kaleidoscopically. If minute slivers of glass could be imagined in the vitreous and the ophthalmoscopic mirror could be reflected on them, no doubt one could get this kaleidoscopic flash and disappearance of the light.

These indications have been so constant in tuberculous cases as to be startlingly significant. Prior to, and along with these appearances, there is a hyperemic disc with filling cup, veins full, dark and beaded. As pointed out by Patterson, the vessels are frequently tortuous.

The vision varies from normal to 20/60, sometimes lower. Our contention is that these phenomena precede the more gross retinal changes such as exudates, tubercle, hemorrhages, etc.

With this brief outline of our subject, we wish to mention briefly some cases that we have examined bearing out the ideas stated above. These patients were in the hands of first class

internists, who assisted us by their study and skill in placing the diagnosis beyond question.

Dr. Kischner referred a Mr. D., age 25, a very large, healthy looking young fellow, who was suffering from ordinary asthenopic symptoms. During the examination for glasses, the fundus was studied and the picture was a striking one. Piled upon the disc in a loose heap were numerous short threads of yellowish white color. Along the vessels, which were distinctly tortuous, were sheath like areas of the same color. Forward to the vessels were highly refractive lines. Further forward, in the vitreous, were many irregular lines that reflected the light in the same way.

It was found that they were using the Kromeyer Lamp for his pulmonary tuberculosis, from which he was suffering in a subacute way. It was thought at first that this might be the cause of his trouble, but he assured us that his eyes were abundantly protected with a heavy black cloth. The very next day, the same doctor referred a ten year old lad, M. B., whose father was on his death bed with pulmonary tuberculosis. The lad himself had enlarged bronchial and cervical glands, but no temperature or other evidence of activity. He had the same asthenopic symptoms. His retina was still more striking in appearance. The superior external quadrant of the nerve and adjacent retina was distinctly swollen, and of a greyish appearance. We do not think that this was an actual tubercle, as described by Finnoff and Jackson, for it did not grow rapidly or tend to absorb, but remained nearly stationary (during the period of observation of about two years). It was one disc diameter in extent. Along each artery and vein was the same pale colored sheath as in the other case. The highly refractive lines over the vessels and in the vitreous were also present. We assume that these make the so-called shot silk appearance, and those in the vitreous the so-called fracture lines. Since the days of the discovery of the ophthalmoscope, these have been observed, and Fuchs in his first American edition warns us that these are normal and not to be confused with actual pathologic processes; also, that they disappear about the fourteenth year.

These cases were examined in Monrovia, California, where the percentage of tuberculosis in children is high.

A large number of children seen in private practice and in the free clinic at Lincoln, Nebraska, who had been studied, and in whom there was no doubt as to their tuberculous condition, exhibited the shot silk appearance, fracture lines in the vitreous, pale colored area along vessels, and varying degrees of change

in the appearance of the disc and blood vessels. The intima of the veins at times seem irregularly thickened, to such an extent in places as to actually interrupt the circulation.

Mr. V. H., a young man of 26. Three years before, he suffered from tuberculous larynx, with ulceration and involvement of both lungs. He had healed nicely and was considered an arrested case. He suddenly developed a severe frontal headache of several days' standing. His physician, thinking it was from frontal sinus disease, referred him for examination. The nose and sinus proved to be normal, so we looked at his fundus. On the disc, standing up around each vessel, but not extending much beyond the disc, was the same straw colored sheath. We told the doctor this would give us a chance to put our ideas to a test—if the exudate meant anything, it indicated a renewal of his process, and in this case probably meningitis. Within two days he developed a Koernig and a Babinski, stiffness of the neck, etc., and within ten days he was dead.

Another exceedingly interesting case was a Mrs. H., a rancher's wife, 36 years of age, who was carried out of the theatre in a state of collapse. When taken home, she was found to be paralyzed on one side. In the right fundus there was a cloud like exudate over the superior temporal vessels. Thanks to the description given by Stark and others, we had no hesitancy in saying that she probably had a small tubercular hemorrhage in the brain. They at first denied any history of tuberculosis, but later confessed that she had had repeated hemorrhages during the past year, and that the X-ray and sputum had also proven positive, though she felt and looked so well, they were unable to believe the testimony of their physician.

Let us call your attention to several well established facts which seem to have a direct bearing on our subject. First, tuberculous infection occurs almost universally during the first fourteen years of life. Second, tuberculosis is largely a blood disease, and it is only natural to think that the first effect would be on the blood vessels themselves, and particularly the veins, because of their slower blood velocity. Third, that tubercle bacilli are found in the blood stream in a high percentage of the acutely active cases, and that their toxins are found in every case where infection exists, otherwise there would be no development of immunity. Fourth, that all old tuberculous exudates are of glistening white or yellowish-white color. Fifth, that tuberculous patients have a low blood pressure; therefore, the swollen appearance of the vessels is not due to increased pressure, but to

a low grade inflammatory process, which causes thickening of the intima and exudation, which later is carried through the lymph channels around the vessels and into the vitreous.

If we are not mistaken, this is in line with the theory advanced by Dr. F. Park Lewis, of Buffalo, that the toxins generated in the blood were responsible for lysis of the vessel walls.

In conclusion, we feel sure that if you will carry in your mind the description of the signs noted above, and refer your patient to a tuberculosis specialist, you will have some startling disclosures where you least expect them. Many times we have been dumbfounded in patients of all ages, to pick up a tubercular history in apparently splendid specimens of humanity. If the patient is not cognizant of his condition, it may be the very earliest evidence of tuberculosis.

DISCUSSION.

DR. F. PARK LEWIS, Buffalo, N. Y.: I have felt with Fuchs, that the condition of the blood vessels which he has found were not abnormalities. It is not impossible, however, that they may be the results of previous tubercular infections which have largely disappeared, or the beginnings of some which may later be manifested.

Until very recently, tubercular retinitis has not been recognized as an ophthalmologic entity. It is not referred to in any of the text books, except incidentally in a footnote by Duane in Fuchs. The reason is, that a fact must be generally accepted before it can be published authoritatively. During the last few years, however, there has grown up in the journals a very extensive literature on this subject.

There are two retinal conditions which can be pretty definitely accepted as having a tubercular origin. One is the recurrent hemorrhages of adolescence. It is still a question whether these hemorrhages are always caused by tubercular infections. Indeed, it is known that they may be caused by toxins of other bacterial forms.

Whitehead stated that never yet had he seen a tubercle of the retina. He had seen what appeared to be a tubercular exudate at the margin of the optic disc. Jackson had an opportunity of observing five cases of retinal tuberculosis extending over a period of two years, and he made most careful observations. In these the veins seemed to be chiefly involved, and the exudate was endothelial in character.

Calmette, in his recent work, says that tuberculosis is not a disease of the lungs, liver or kidneys, but of the lymphatics, and that the tubercle bacillus or toxin is carried into the blood current through the lymphatics or into the blood vessels.

From Zentmayer's admirable article on Tuberculosis of the Retina, reviewing Eales' cases of retinal hemorrhage, described years ago, it would seem that in all of them were the physical depression and lowered blood pressure suggesting a tubercular origin.

Whether or not the conditions described by Teal are verified, they will be of value in directing our attention to the eyeground when a pos-

sible tubercular condition may be suspected, and in such it would be advisable to make an early tubercular test, and to take the morning and evening temperature.

DR. JAMES A. PATTERSON, Colorado Springs, Colo.: In 1922, I presented this subject to the Colorado Congress. I had then studied 325 cases from private practice, and I have continued to watch this characteristic of multiplicity and undue tortuosity of the retinal vessels. Of course I practice in a neighborhood where a great many tubercular people and their progeny are seen. The astonishing thing to me is how you find this condition in children whom you know nothing about, yet on inquiry a tubercular family history is frequently disclosed. It is also noticed in adults with an active or stayed tuberculosis. I have never known these children to develop tubercular ocular lesions later.

DR. ROBERT SCOTT LAMB, Washington, D. C.: The question I believe is possibly a little bigger and broader than Dr. Teal has pointed out. We see these cases, and unquestionably they make a very good soil on which tuberculosis may start. But let us consider for a moment the fact that tuberculous individuals are often neuropaths, many of them have a definite vagotonia, in which you find tortuosity of the vessels and marked venous congestion. This condition is very common, and often antecedes a condition in which the blood vessels have linear fibrous changes, and if you are going to point that out as a cause of tuberculosis, as a definite forerunner, I think you might consider it is a definite forerunner of a lot of other things that occur in the eyes later in life, even disseminated choroiditis of circumscribed type, and hundreds of cases of choroiditis due to eyestrain or to direct injury.

DR. EDWARD JACKSON, Denver, Colo.: This subject of retinal tuberculosis, or tuberculosis in other parts of the eye, being an important one, and having myself for many years failed to recognize its importance, or its frequency, makes me appreciate that it should be brought repeatedly before bodies like this from different points of view, until there is a more general appreciation of the number of these cases that actually occur.

My own view of tuberculosis involving the eye I have tried to express in my paper entitled "Tuberculosis as a Focal Infection." It is an infection, and one to which human beings seem to be particularly liable. The statistics that were drawn years ago from autopsies, in Paris, particularly, and in other places, seem to show that a very large majority of those who die from all causes have been, at some time, affected by tuberculosis. These statistics are more and more reinforced by our wider knowledge of the subject. Now, if that is the case, if a large proportion of human beings will at some time in their lives be affected by tuberculosis in some way, any study based on a large number of eyes, even if not strictly statistical, must take that fact into account. Say that nine-tenths of the people are at some time tuberculous. If all have retinal reflexes, all who have tuberculosis will show such reflexes. All persons are liable to alteration in the appearance of the vessels under eyestrain, as Dr. Wm. F. Norris used to emphasize, and Dr. Risley also. The appearance of the vessels in cases of eyestrain should be considered even if the patient has tuberculosis. Statistics of this sort are useless

unless we have statistics of an equal number of cases in which these appearances were absent, or less frequent.

With reference to the appearance and size of the retinal vessels, we all realize that they vary very widely. My attention was directed to this many years ago, when I began to observe the condition of the vessels of the retina, and note where there was a striking case of tortuosity or there was a more liberal supply of blood to the retina than the average. But I cannot find, by going back over my records since my attention has been brought to this, that the tortuosity of the vessels can be taken as a sign of tuberculosis. The same is true of the reflexes in the retina. Their frequency first struck me when I began to use the ophthalmoscope on eyes of school children with Dr. Risley. As Fuchs says, they are much more frequent in children, although we find them in adults. Other things must be taken into account before we draw any conclusions from retinal reflexes. One is the size of the pupil. You will see, as I have, very striking reflexes before the pupil is dilated, and afterwards they are all gone. Those who have been working with a red free light know how much more striking the reflexes are with this light than with other methods of examination.

Another point is that a small source of light multiplies these reflexes, and if you get a strong light like sunlight and use it from a small point, they are still further exaggerated. Working with a new ophthalmoscope that has a different sight hole in the mirror, is likely to exaggerate the frequency of things like this.

I cannot but think that some of these things that have apparently been connected up with tuberculosis, really have nothing more to do with it than the color of the eyes or the hair have to do with whether a man will be run over by an automobile. Very extensive statistical studies would be required before I would be convinced to the contrary.

DR. FREDERICK F. TEAL, Lincoln, Neb.: I still think, in spite of Dr. Jackson's remarks, that there is some connection between the fact that nine-tenths of the people have tuberculosis, and nine-tenths show this condition which we have outlined, particularly in children. The fact that children usually recover from their signs of tuberculosis near the age of fourteen, connected with the fact that we do not see these cases nearly so much after patients are fourteen, seems significant to us.

Dr. Jackson brought out the fact about the pupil. I am glad he spoke of that. If you try to observe these conditions through a dilated pupil, you will probably not find them.

I am disappointed that no mention was made of the appearance of the exudate along the vessels. That is a condition that in some cases is most striking, and the thing that appeals to us is, that after the cases are arrested, that condition disappears.

GLAUCOMA AFTER CATARACT EXTRACTION.

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NEW YORK CITY.

In the consideration of this subject, it has been the endeavor of the writer to exclude all cases in which hypertension existed before the extraction of cataract, and to omit those cases in which hypertension develops after the needling of juvenile cataract, or of senile cataract to hasten maturity.

It has been the practice of the writer to test the tension of all eyes of patients by palpation, and if there is any suspicion of hypertension, to measure it by means of the tonometer, using the Schiötz instrument. The occurrence of glaucoma after cataract extraction was recognized many years ago. Bowman¹ writes: "Such glaucomatous tension is particularly apt to come on after needle operations. . . . The glaucomatous hardness is by no means an indication or a result of mere iritis. . . . Like ordinary idiopathic glaucoma, this glaucomatous state may commence without inflammation and remain long without pain, although it is far more commonly accompanied by both. It may come on twenty-four hours after the needle has been used, but usually on or after the second day." Mr. Bowman proceeds to describe the development of partial or complete iris bombé after cataract extraction, and indicates that relief may be obtained by puncturing the bulging iris with a broad needle.

FREQUENCY OF THE OCCURRENCE OF GLAUCOMA AFTER CATARACT EXTRACTION.

E. Treacher Collins² writes: "From the commencement of 1885 to the middle of 1889, there were at the Moorefields Hospital 1,405 senile cataracts extracted; of these, nine were lost from glaucoma, or 0.64%." This percentage represents the very severe cases.

The late Dr. Herman Knapp³ states that glaucoma occurs in at least one per cent after needling, and in about the same percentage when needling has not been performed. I was closely associated with Dr. Knapp for eight and a half years. Cases of glaucoma after cataract were not uncommon in his practice. He was then of the impression that the total percentage was about three per cent.

A. von Hippel⁴ reports two cases in 184 extractions.

Gama Pinto⁵ reports six cases in 326 extractions, a little less than two per cent.

Blaskowitz⁶ reports two cases in 300 extractions.

Graefe⁷, in 1869, found that glaucoma was not rare after extraction or discission.

While I have no exact records, I am firmly of the opinion that glaucoma after cataract extractions has occurred in at least $1\frac{1}{2}\%$ of the cases of senile cataract operated on by me, but in not more than $1/10$ of 1% after discission of capsular cataract, and in not more than two cases have I found it necessary to remove the globe for absolute glaucoma.

METHODS OF EXTRACTION FOLLOWED BY GLAUCOMA.

It is the consensus of opinion that glaucoma may develop after any of the known methods of extraction. "Natanson⁸ collated and analyzed the records of thirty-seven cases. They show that immunity from subsequent glaucomatous complication is not ensured by any particular method of operation, even by extraction in the capsule." (Priestly-Smith.)

Rumschewitsch⁹ reports three cases of glaucoma after cataract extraction, in one of which the lens was removed in its capsule.

Priestly-Smith¹⁰ writes: "Glaucoma after cataract extraction may occur during the after treatment, or after recovery is apparently complete, or even after a good result is maintained for years; it is not absolutely unavoidable by any particular method of operating."

H. Gifford¹¹ reports six cases of glaucoma occurring in eyes in which partial or complete spontaneous absorption of the cataract had taken place—cases in which hypertension had not previously developed.

TIME OF INCIDENCE.

Hypertension may develop in twenty-four to forty-eight hours after extraction, and within a few hours after needling; it may develop many years after the extraction. Rudel¹² reports three cases, in one of which "simple glaucoma" developed six years after cataract was removed from both eyes. Ayres¹³ reports one case in which glaucoma developed in one eye seven years, and in the fellow eye 20 years after extraction with iridectomy. Pagenstecher¹⁴ states that the hypertension usually follows in the first few days. Such eyes may never have shown hypertension, but glaucoma after cataract extraction is much more frequent in eyes that have shown hypertension.

Lichtenburg¹⁵ reports a case of simple extraction O. S., in which discission was done six months later. Resulting vision 20/20—. Five years later, cause for operation on the fellow eye. The vision of the operated eye was then 20/50. Tension 40 (Schiotz). Iris bombé. The tension was permanently relieved by passing a narrow Graefe knife through the anterior chamber in the horizontal meridian from limbus to limbus, piercing the iris and permitting of free passage between posterior and anterior chamber. (Operation described in Meller's Surgery of the Eye.)

In cases of the development of an epithelial lining to the surface of the tissues of the anterior chamber, the time that elapses before hypertension develops may be three to nine months.

W. Ralston and E. L. Goar¹⁶ report four cases of glaucoma after cataract extraction. In case three, simple extraction on both eyes was performed, the last about three weeks after the first, without complication. Healing uneventful. Vision with correction O. D. 20/30+, O. S. 20/20. Glaucoma developed in the right eye five years later. Iridectomy in that eye was followed by hemorrhage into the vitreous body. Resulting vision, fingers at three feet. O. S. was not affected.

CAUSES.

The causes of glaucoma after cataract extraction are quite numerous:

1. Gifford¹¹ believes that the spontaneous absorption of the cortex of a senile cataract produces, in some cases, an increase of tension, which may be temporary, or which may lead to complete blindness. He had observed ten cases of almost complete absorption of senile cataract, six of which were complicated with glaucoma. He recommends that senile cataract should be kept under observation and operated before it becomes too mature, even if the vision of the other eye is still good.

2. The ingrowth of epithelium through a corneal incision into the anterior chamber, where in some cases it may line the entire anterior chamber, closing the lymph channels through which the intraocular fluids normally escape, a condition spoken of by Collins as epithelial cyst of the anterior chambers. The author has examined two such cases microscopically. The eyes were removed because of absolute glaucoma accompanied by pain. The condition was not diagnosed, so far as the epithelial lining of the anterior chamber was concerned, until the eye came to section. The diagnosis of this condition is almost, if not quite,

impossible before the eye comes to section, because of the transparency of the epithelium. (For an illustration of this condition see Fuchs, 5th Am. Edition, p. 445.)

3. The incarceration of, and in a few cases the prolapse of, iris and lens capsule. Natanson⁸ collected and analyzed the records of 37 cases. He states, according to Priestley-Smith, that "They show that, in the majority of the cases, there was some visible complication involving the iris, or the capsule, or both. . . In some cases the eye appeared to be quite free from any complication of the kind. This negative evidence is, however, not quite conclusive, for slight adhesion of the kind in question may be undiscoverable in the living eye." "Becker examined with the microscope 38 eyes from which cataract had been extracted, and in only 1/3 of these was the iris free from the scar, although 32 of the 38 eyes were removed, not on account of any trouble during life, but after the death of the patient. He expressly notes that minute adhesions of the iris or capsule with the scar may be quite invisible in the living eye."

Parsons¹⁷ writes: "From a review of the cases reported, it would seem that the common feature present in all is incarceration of capsule, or iris, or both."

It may lead to no evil results. It may cause increased tension by:

1st, Setting up severe iridocyclitis.

2nd, By causing sufficient blocking of the angle to bring about hypertension.

3rd, Incarceration of lens capsule.

4th, Advancement of vitreous body into the anterior chamber. In this connection, we may refer to the very excellent and widely known paper by Mr. E. Treacher Collins¹⁸, in which he describes the microscopic findings in ten eyes removed for glaucoma after cataract extraction. The following is a quotation from that article:

"In nine of the cases, the cataract was senile; in one, probably traumatic. In five, a cataract had been successfully removed from the patients' eye without any symptoms of glaucoma having manifested themselves. In three, the glaucoma came on after a needle operation. In three others, it came on with iritis accompanied by keratitis punctata, and the second eye in each of these cases became affected with sympathetic ophthalmitis. In the remaining four, slight iritis, only, followed on the operation; in one of them, however, the patient knocked her eye on the tenth day and reopened the wound.

In one case, a preliminary iridectomy, and in eight of the cases an iridectomy at the time of the extraction, was performed. The capsules were opened in different ways; in some, the face being lacerated, in others a peripheral incision, only, being made. In two, a portion of the anterior capsule was torn away with the forceps. In one, the capsule was entirely removed.

Pathologic examination revealed in all the cases, except this last, adhesion of the lens capsule to the extraction cicatrix; and in this last one, the hyaloid of the vitreous had come forwards and adhered to the corneal scar.

In the three cases which accompanied keratoiritis, and in the case in which the corneal wound was reopened by a blow, the capsule had become converted into a thick membrane by inflammatory plastic effusion, and the lower part of the iris was adherent to it in its whole length.

In the other cases, no such thickening had occurred, and the adhesions of the iris were confined to the pupillary margin. In all the cases, with the exception of the one in which the lens was removed in its capsule, some cortical matter had been left in the peripheral portions.

The angle of the anterior chamber in the coloboma was in all the cases blocked either by the root of the iris which had been left, or by the foremost of the ciliary process, which had become dragged forwards and held in close contact with the posterior surface of the cornea by the upper portion of the entangled capsule. The angle of the anterior chamber elsewhere was also blocked by peripheral adhesion of the root of the iris, in all except the keratoiritis cases. In these three, there was inflammatory cell infiltration of the tissue of the iris and of the meshes of the ligamentum pectinatum, also collections of round cells in small clusters on the posterior surface of Descemet's membrane. The ciliary processes were in most of the cases elongated forwards, and at the upper part of the eyes flattened by the pressure from the fibers of the suspensory ligament. The choroid in one case was much thickened by inflammatory infiltration; in two, there were patches of dilated vessels, and in two of the others it was atrophied. The optic nerve was cupped, and showed glaucomatous changes in all except the three cases accompanied by keratoiritis; in these, it was slightly swollen and slightly infiltrated by small round cells. The vitreous in four cases had the remains of small hemorrhages in it; in all the cases except one, its consistency was thinner than normal; in some it was detached posteriorly from the retina.

In the cases in which the glaucoma came on after needling, the adhesion of capsule to the extraction scar must have existed since the removal of the cataract, but the increased tension did not occur until after dissection of the capsule had been performed, in one case not until after the second dissection, and three and a half years subsequently to the operation. Microscopic examination of the cornea in this case showed two scars with attachment of lens capsule to them. The second needling had possibly caused a fresh adhesion and still further advance in the position of the capsule, and consequently of the iris. In another case, a small prolapse of the vitreous through the needle puncture in the cornea was observed."

Stoelling¹⁹ reports a case in which a permanent good result was obtained by a modified von Graefe operation on O.S. Later, the right eye was operated on by the same method. Anterior chamber restored only after ten days. Hypertension on the 14th day. Eight operations were performed for the relief of tension in 16 weeks. Eye removed. Incarceration of iris and capsule throughout the entire extent of the wound was found.

5th. Retention of cortical lens matter. This is a well recognized cause of hypertension, probably because of changes in the aqueous humor, making it more difficult for the fluid to pass through the lymph spaces, as well as because of obstruction due to lens fragments in the anterior chamber and encroachment of the root of the iris on the iris angle, because of increased pressure behind the iris.

Risley²⁰, writing of glaucoma appearing after needling of cataract, states that overloading or blocking of the filtration angle may take place after capsulotomy following extraction of hard cataract, not only by setting free cortical masses entangled in meshes of the capsule, but by the presence of semifluid vitreous which escapes into the anterior chamber through a rent in the posterior capsule caused by the operation. He reports two cases.

6th. Retinal or choroidal hemorrhages. The effect of hemorrhage into the retina in the production of glaucoma in an aphakic eye is illustrated by the following cases:

May 2, 1918, Mr. P.S., age 55 years. Nuclear incipient cataract O.D. Tension normal by palpation.

December, 1920, double preliminary iridectomy.

May 11, 1922. Extracted cataract O.D., Incision at limbus. Short conjunctival flap. Healing uneventful. June 5, 1922, vision with glasses 20/40+.

October 24, 1922, extracted cataract O.S. Operation as for

O.D. Discission capsule of O.D. Healing both uneventful. April 9, 1923, vision of each eye 20/20—. Tension normal by palpation. May 7, 1923; during the last two weeks has not seen so well with the right eye. Ophthalmoscopic examination discloses tortuous vessels, particularly the veins. Some arterial sclerosis. Numerous small retinal hemorrhages, particularly about the macula. Cornea clear. Tension O.D. 33; O.S. 18. History of mild intestinal disturbance about three weeks ago. All tests now negative. Blood pressure within the normal range. prescribed miotics.

May 14, 1923; tension O.D. 20. Relative central scotoma. V. 20/100. T.O.S. 18. May 28, 1923, T.O.D. 47, T.O.S. 18. June 5, 1923; T.O.D. 18. July 19, 1923; T.O.D. 30, T.O.S. 18. July 26, 1923; T.O.D. 20, T.O.S. 18, since which time the tension has remained normal. Aug. 2, 1923; discission capsule O.S. Aug. 9, 1923; vision with glasses O.D. 6/200, O.S. 20/20. No visible incarceration of iris or capsule.

April 4, 1919, Mr. J.V.B., age 62 years. Cataract O.S., extracted by a colleague in 1911. Eye lost some years later by secondary infection of a cystoid scar. Mature uncomplicated cataract O.D. Tension normal by palpation. July 31, 1919; extraction cataract O.D., combined method. Narrow conjunctival flap. Healing uneventful.

August 18, 1919; vision with glasses 20/30. April 3, 1923; vision 20/30. June 15, 1923; hemorrhagic retinitis. Tension normal (palpation). Blood pressure 168 (systole). Has an enlarged prostate and a mild colitis. Aug. 7, 1923; hemorrhagic retinitis more marked. Tension plus. Cornea clear. Anterior chamber deep. Advised the use of miotics and consulted with family physician. Aug. 23, 1923; T.O.D. 65 (Schiotz). From August 23, to September 20, 1923, tension varied from 40 to 70. Vision 3/200. September 20, 1923; trephining, using a 1½ mm trephine. Healing uneventful.

7th. Iritis and cyclitis.

8th. Adhesion of iris to remaining lens capsule, causing the formation of partial or complete iris bombé.

These two conditions are well set forth by Priestley Smith²¹, who writes: "In some cases, the iris and posterior capsule, being united and coated by inflammatory exudation, appear to form an impermeable or insufficiently permeable diaphragm across the eye, which checks the passage of fluid from the ciliary processes into the aqueous chamber. An excess of fluid becomes imprisoned behind this diaphragm. This may happen although a good iri-

dectomy has been made. In a case of this kind, on the eighth day after extraction and in presence of acute iritis, with free exudation into the anterior chamber and very high tension, which had twice rapidly returned after paracentesis of the aqueous chamber, I made an iridectomy downwards, tearing completely through the adherent membranes and obtaining for the moment a jet black pupil. The eye recovered 'permanent' normal tension and good vision".

In some cases, having a pupillary area sufficiently clear to admit of good vision, the adhesion of the iris to the remaining capsule may be sufficient to shut off the area of pupil and coloboma, and to extend along the posterior surface of the iris in places to the ciliary attachment, preventing the free passage of fluids from the posterior to the anterior chamber. Hypertension results. The iris is bulged forward over the areas free from adhesion, forming what appears to be multiple cysts—a partial iris bombé. This condition, which follows a mild iritis, develops some weeks or months after extraction. The hypertension can be completely relieved by an iridectomy which includes a portion of the bulging iris. A case of this nature occurred in a woman of advanced adult life, within the last year, who was under the observation of Dr. John Wheeler and myself. An iridectomy made as suggested above gave complete relief, after paracentesis and dissection of capsule had failed.

9th. Foci of infection located in teeth, paranasal cavities or tonsils, increasing tension by setting up a low grade iridocyclitis²².

10th. Cases without known cause.

January 14, 1899, Mrs. M.F., age 54. Incipient nuclear cataract O.U. Tension normal by palpation.

December 14, 1900. Removed cataract O.D. by simple extraction. No incarceration of iris or capsule. Healing uneventful. Resulting vision six weeks later 20/20—.

July 18, 1902. Eye in good condition V = 20/20 —.

August 20, 1902; attack of acute glaucoma, severe pain, nausea. As I was absent from the city, my assistant consulted with Dr. Herman Knapp, who performed iridectomy on September 19, 1902, as the glaucoma was not controlled by miotics.

Feb. 29, 1908; tension of O.D. about normal since the iridectomy was performed in 1902. V = 20/30 +. Slight contraction of nasal part of field. March 12, 1908; extraction of cataract O.S. by the combined method. Healing uneventful.

Nov. 26, 1912; tension O.U. normal (palpation). V. O.D. 20/30; O.S. 20/20 —.

Mitchell²³ reports two cases of simple extraction. Healing uneventful. In case 1, dissection of capsule two weeks later. In case 2, dissection one month later. No reaction. Acute glaucoma followed two months later in the first, two weeks later in the second case. Thought that the iris was adherent to capsule in the first case. Attempted iridectomy. Lost much vitreous. Wound healed. Tension became normal and remained so. Ultimate V. = L.P. In the second case, a free iridectomy under general anesthesia resulted in reducing tension to normal. Ultimate vision of 20/40.

Bulson²⁴ reports two cases:

1st case: Male 65 years. Combined extraction. Healing uneventful. No evidence of iritis. No entanglement of iris, capsule or vitreous body. Obtained vision 20/30. Four weeks later, acute glaucoma. Three months later, enucleation for absolute glaucoma. The microscopic examination showed no entanglement of iris or capsule. Iris angle unobstructed.

2nd case: Similar to case one. The microscopic examination disclosed evidence of a "serous cyclitis". No entanglement of iris or capsule. It is thought that the inflamed ciliary processes poured into aqueous chamber a serous fluid, which escaped from the eye with greater difficulty than the normal secretion and clogged the filtration angle." Bulson does not place great value on the influence of incarceration of iris, etc. in the production of secondary glaucoma.

TREATMENT.

In all cases, necessary systemic treatment should be advised. The local treatment to be employed depends of course largely on the cause of the secondary glaucoma. If the case is one of iridocyclitis, miotics may suffice to give relief from tension until inflammatory products that cannot escape through the filtration process has disappeared, or one or a number of paracenteses may suffice to carry the patient over the period of the production of inflammatory products that cannot escape through the filtration angle. In quite a high percentage of these cases, the treatment mentioned will suffice. There are a few cases of moderate increase in tension, in which there is no visible evidence of iritis or cyclitis, that will eventually recover by the instillation of a solution of pilocarpin continued for some months. In cases of hypertension due to retinal or choroidal hemorrhages, proper systemic treatment, plus the use of miotics locally, will suffice in some cases. In a recent case in which the tension ranged from

40 to 75 (Schiötz), and so continued until it caused a deterioration of vision, I resorted to trephining.

I would advise operation only as a very last resort.

In glaucoma after cataract extraction with incarceration of capsule and iris, if the tension cannot be kept at or below 28 (Schiötz) by the use of miotics and massage, we may sometimes obtain relief by dividing the bands of capsule and relieving tension over the iris by snipping the columns of the coloboma, using the de Wecker scissors. In a few cases, this procedure has given good results in my experience.

In cases with a free iris angle, iridectomy may be sufficient. This operation is particularly effective in cases of glaucoma following simple extraction. The following are cases in point:

H. Knapp²⁵ reports ten cases of glaucoma—nine after simple extraction, one after a combined extraction. All followed discissions of the capsule in from 12 hours to six months. The Knapp knife needle was employed in all. Of these cases, one was blind when seen two and one-half years after the discission. Two were apparently cured by the use of miotics, and seven were cured by iridectomy.

Risley (l.c.) reported two cases. One was cured by iridectomy and one by the use of miotics.

In the greater number of cases, an operative procedure having for its purpose the forming of a filtering cicatrix, will be advisable. This will be the better procedure in all cases in which the filtration angle is permanently blocked other than by traction bands. In a few cases, I have obtained fair results by trephining over the iris coloboma. In trephining or operating after the Lagrange method, it is sometimes difficult to seize the iris with forceps. In such cases, the sharp hook of Tyrell is very serviceable.

Finally: Enucleation may be necessary. This is particularly indicated in those cases with incarceration or prolapse of iris or capsule with iridocyclitis, which may possibly lead to sympathetic inflammation.

PROGNOSIS.

The prognosis must always be guarded, both as regards loss of vision and removal of the eyeball.

Dalton²⁶ divides his reported cases into three groups: 1st, after simple extraction; 2nd, after combined extraction; 3rd, after discission of secondary cataract. In nearly all of the cases reported, there was some complication: Iris or capsule adherent to

wound; lens cortex retained; iridocyclitis, etc. In all of the cases, with but one or possibly two exceptions, the result was blindness. A number required enucleation.

In Knapp's cases referred to above, there were very few complications. One only became blind.

Albrand²⁷ reports one case following discission of secondary cataract cured by paracentesis.

Ayres¹³ reports five cases. In the first case, vision was virtually lost in both eyes after combined extraction. In one case, vision was lost in one eye. The fellow eye was operated on in the same manner (combined extraction). Glaucoma did not develop in the second eye. Enucleation was necessary in one case in which iridectomy was unsuccessful. Two were apparently cured by miotics and paracentesis.

Of two cases reported by Risley, both recovered useful vision.

In an experience of approximately twenty-five cases, the writer has been obliged to remove the eyeball in not more than three. Apparently thirty per cent have not required operative procedure, and vision of all degrees of acuteness, from 20/20—to fingers at a few feet, has been secured and retained.

SUMMARY.

1. Glaucoma, either acute or chronic may occur after any form of cataract extraction, or after discission of the capsule, in eyes that are not otherwise glaucomatous.

The history of some of the cases indicates that chronic simple glaucoma may develop in some eyes after cataract extraction, without having the operation as a causative factor.

2. The frequency of incidence is approximately one and three-fourth per cent. Discission of the capsule is followed by glaucoma about as frequently as is the primary operation.

3. The ingrowing of epithelium occurs only in those cases in which the incision is made in the cornea, and is most frequent in corneal incisions that are slow to close.

4. The individual case must determine the treatment to be employed in that case. Glaucoma after cataract extraction, if of a mild type, may recover spontaneously. Miotics will suffice to bring about recovery in a small percentage of the cases, notably in mild cases of iridocyclitis and in some cases of hemorrhagic retinitis. Paracentesis, iridectomy, the Lagrange operation, trephining and enucleation are operations that are occasionally indicated.

5. Prognosis: Removal of the eyeball is necessary in some

cases, but vision ranging from perception of light to 20/20 may be retained in a high percentage of the cases.

6. In operating for the extraction of cataract, with a desire to avoid glaucoma, it is advisable to place the incision in vascular tissue, and to have the resulting wound free from incarceration of iris or capsule.

In performing discission of secondary cataract, the knife needle should not pierce the cornea if entrance through the cornea can be avoided.

BIBLIOGRAPHY.

1. Bowman. Roy. Lond. Ophth. Hospital Reports, 1865, v. 4 p. 365.
2. Collins, E. Treacher. Trans. Ophth. Soc. U. K., v. X, p. 174.
3. Knapp, H. Norris and Oliver System, v. III, p. 786.
4. v. Hippel, A. Arch. of Ophth., v. XLIV, 2, p. 387.
5. Gama Pinto. Klin. Monatsbl. f. Augenh., 1896, p. 295.
6. Blaskowitz. Zeitsch. f. Augenh., v. XIV, p. 345.
7. Graefe. Parsons' Pathology, v. III, p. 108.
8. Natanson. Ueber Glaucoma in aphakischen Augen, Dorpat, 1889.
9. Rumschewitsch. Zehender's Monatsbl., June, 1896.
10. Priestley-Smith. Norris and Oliver's System, v. III, p. 637.
11. Gifford, H. Amer. Jour. of Ophth., v. I, p. 83.
12. Rudel. Ueber Glaucom. Ber. u. d. Augenklinik d. Wiener Universität, Vienna, p. 132.
13. Ayres. Arch. of Ophth., 1897, v. 26, p. 208.
14. Pagenstecher. Zehender's Klin. Monatsbl. f. Augenh., May, 1895.
15. Lichtenburg. Jour. Mo. St. Med. Assn., v. XIV, p. 345.
16. Ralston, W., and Goar, E. L. South. Med. Jour., v. XV, p. 581.
17. Parsons. Pathology, v. III, p. 108.
18. Collins, E. Treacher. Trans. Ophth. Soc. U. K., v. X, p. 108.
19. Stoelling. Arch. f. Ophth., v. XXXIII, p. 177.
20. Risley. Ophthalmology, 1909-1910, p. 572.
21. Priestley-Smith. The Pathology and Treatment of Glaucoma, London, 1891, p. 57.
22. Hinsdale and Key. Arch. of Ophth., 1922, v. LI, p. 507.
23. Mitchell. Ophthalmic Record, 1897, v. VI, p. 355.
24. Bulson. Trans. A. Acad. O. and O-L., 1907, p. 236.
25. Knapp, H. Arch. of Ophth., 1892, v. XXI, p. 293.
26. Dalten. Mitt. a. d. Augenklinik, Stockholm, 1901, H. 3, p. 75.
27. Albrand. Arch. f. Augenheilk., v. XXXIII, p. 208.

CATARACT AFTER GLAUCOMA. ITS ETIOLOGY AND TREATMENT.

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That opacities of the lens develop rather frequently in glaucomatous eyes is known by all close observers of large experience. Fortunately, only occasionally does the process go on to the development of complete cataract. The problem of the proper handling of chronic simple glaucoma is difficult enough, and it has had free discussion, but the serious complication of cataract development after the existence of glaucoma has had astonishingly scant discussion. The extreme seriousness of the condition is evident, and we should face it squarely, unpleasant as it is.

So little is known of the process of cataract formation, even of the primary type, that we are kept within the domains of speculation, but hope of prevention resides in the study of causes of cataract.

The old notion of glaucomatous cataract was that it was characteristically greenish in color, but this was largely due to a curious deception. Vogt¹, in his slit lamp corneal microscope studies, has not discovered any characteristics peculiar to glaucoma cataract. Various types develop, but it may be said that opacity in glaucomatous cataract, as in senile cataract, is likely to start around the equator of the lens, where the cortical fibers are youngest and most sensitive to unfavorable influences, and along the suture lines of the fibers. Lens degeneration which does not cause opacity in the visual line may be of no importance in glaucoma, and our hope is always that cataract will not become a serious factor in impairing the sight in glaucoma.

Etiology. Let us first consider the development of cataract in glaucoma independent of operation. One of the most evident things about glaucoma, is that there is *interference with the normal secretion and circulation of the aqueous*. Partial blocking of the drainage angle hampers the outflow from the anterior chamber, and increased intraocular pressure hampers the secretion of aqueous by the glands of the ciliary body. The lens receives its nourishment from the aqueous, probably by osmosis, and interference with secretion and outflow of aqueous results in slowing down of lens metabolism, sometimes to the point of lens fiber degeneration.

General toxic conditions may be causative. In dealing with the etiology of cataract, Kirkpatrick² stresses the probable importance of disturbance of the endocrine glands. Cataract has been known to follow thyroidectomy³. It is more than likely that disturbance of function of the endocrine glands, with their curious interdependence, may play a part in the causation of many cases of glaucoma. And it should be borne in mind that such disturbance may be contributory in the development of, first, glaucoma, and subsequently cataract. The nutritive process of the lens is one of slow action, and degeneration of lens cells is likewise slow.

General toxicity from hookworm, pellagra, diabetes, gastrointestinal disease, focal infections and from many other causes has been held accountable for cataract. Until we know more of the incipency of glaucoma, we may well reckon with the possibility that general toxicity may in some cases stimulate the aqueous glands of the ciliary body to excessive secretion, and so have to do with the origin of glaucoma, as well as of cataract. In such cases, the rise of tension would precede the degeneration of the lens cells.

We can no longer doubt that *glare* is a factor in the production of cataract. Glass blowers cataract is recognized⁴. Healy⁵ found lens opacities in about 40% of tin plate millmen of 35 years and older. Van der Hoeve⁶ calls attention to interesting statistics by Frederick Gross, which show that where woman work in the fields with the men, and so are equally exposed to glare, they develop cataracts in numbers about equal to the men; while where women do not work in the fields, they develop far fewer cataracts than the men so exposed.

A few weeks ago, in Vogt's laboratory in Zurich, the staff was astonished by the development of a cataract in the eye of a rabbit from 15 minutes exposure to infrared rays. So it may not be absurd for us to consider the possibility that the moderately dilated pupil in neglected glaucoma, in the presence of other factors, may aid in the production of cataract through lack of control of the admission of glare. Likewise, may it not be possible that a large coloboma following iridectomy may have an influence? If so, we have an argument against the wide iridectomy and in favor of the buttonhole iridectomy of corneoscleral trephining.

Then hereditary *predisposition* may be a factor. Any glaucoma patient may be predisposed to cataract, and in such a patient, hypertension would hasten the cataractous process.

Another contributory cause of cataract may be found in the *unfavorable effect of the glaucoma on the general condition of*

the patient. Justifiable anxiety and apprehension are quite common among glaucoma patients, and a genuine lowering of vitality sometimes results. Lens degeneration may be one manifestation of the depressed general state.

Cataract after Operation for Glaucoma. Direct injury of the lens has occurred too frequently in operations for glaucoma, particularly in iridectomy. The anterior capsule may be injured by the keratome, iris forceps or spatula. In inserting a keratome, it is well for the surgeon to make sure that the point of the instrument enters the anterior chamber well back in the iris angle, and that its direction is kept toward the posterior surface of the cornea. If injury must be, it is far better for the surgeon to injure the endothelial lining of the cornea, or Descemet's membrane, or even the substance proper of the cornea, than to cause a cataract by traumatism to the lens. Unfortunately, text book instruction for the operator to grasp the iris at its pupillary border is in part responsible for cataracts following iridectomy. The surgeon should keep the points of the iris forceps at least a millimeter away from the margin of the pupil in performing iridectomy. Finally, in using the spatula, this instrument should not be used to stroke the iris within the aqueous chamber. It should be used only to disengage the iris from the wound.

Iridocyclitis following operation for glaucoma may set up opacity of the lens. Inflammation of the iris and ciliary body are especially likely to occur if iris tissue is left in the wound. General conditions which may be held in part responsible for the glaucoma may contribute in the causation of iridocyclitis following operation, which in turn may be causative of lens opacity.

Again, persistent hypotony, resulting from operation for glaucoma, may have cataract as one of its degenerative manifestations. Serious hypotony from operation is rare, but it is most likely to follow trephining.

Treatment. Obviously, the glaucoma patient should not be subjected to known possible causes of cataract. Control of intraocular tension, removal of general toxicity, and every aid for the physical condition, protection from glare and all reasonable encouragement and protection against fear of blindness should be offered to the patient by his oculist.

In no case of glaucoma should extraction of a lens be performed in one eye if useful vision exists in the other. So much is at stake, and the outcome is so uncertain, that unless there is serious clouding of the lenses, removal should not be attempted; but if there is practical blindness, or if blindness is imminent, a

patient should not be deprived of his chance for sight on account of fear or timidity on the part of the surgeon.

In case operation is necessary, let me suggest two essentials for expectancy of a favorable outcome. First, a continued reduction of tension by operation to 30 mm. or less (Schiötz) for a period of a month or more. Second, subconjunctival drainage through a filtering scar, manifested by definite edema.

Anything short of definite suggestions at this point would be unsatisfactory. So, without feeling entirely confident, I am going to suggest without evasion. In glaucoma cases, where cataract has developed but where light perception remains, operate rather than consign a patient to inevitable absolute blindness, even if there is serious contraction of the field. If there is hypertension without myotics, first, with a 2 mm. trephine, perform corneoscleral trephining below (in healthy iris if possible), so that the drainage will not be interfered with by subsequent operation. Repeat if necessary. To quote Morax⁷, "we must not despair of obtaining a reduction of tension in a patient on whom a first sclerectoiridectomy has not given the desired result". Secure manifest subconjunctival drainage and persistent lowering of tension to 30 mm. (Schiötz tonometer) or less for a period of at least a month. Then perform preliminary iridectomy or Lagrange above. After an interim of at least a month, extract the lens, making a limbus incision and a conjunctival flap.

If the cataract is not entirely ripe, and if the surgeon is experienced and skilled in the intracapsular extraction, it may be wise for him to remove the lens in its capsule, and so save the eye from the possibility of irritation from cortical remains. Most surgeons would choose the capsulotomy method even for the immature cataract. The incision should be large and well back, with a good conjunctival flap. The capsulotomy, or capsulectomy, should be generous. Other essentials are careful tilting of the lens, and deliberate, painstaking care to avoid separating cortex from the nucleus thus unnecessarily leaving sticky lens material behind and thorough removal of any cortical remains by irrigation. One should not hesitate to use atropin after the extraction, if there is good subconjunctival aqueous drainage.

Elliot⁸ has given the subject in hand much thought, and he suggests corneoscleral trephining above, and later extraction of the lens through a corneal incision to avoid cutting into the drainage area. Elliot's judgment is superior, but I cannot willingly give up the limbus incision with the conjunctival flap.

It may be in place to add, that unwittingly surgeons have performed many cataract extractions on glaucomatous eyes, and that through intraocular hemorrhage, loss of vitreous or post-operative intractable glaucoma, complete blindness has resulted. As Fromaget⁹ has said "thanks to the tonometer, we do not have to expose ourselves to operating for cataract in the presence of hypertension". A good rule for us to follow is to estimate the intraocular tension of cataractous eyes both by the fingers and by the tonometer, and so make sure to avoid ignorantly operating in glaucomatous cataract.

REFERENCES.

1. Vogt, Alfred. Personal Communication.
2. Kirkpatrick, Henry. The Etiology of Primary Cataract. *British Medical Journal*, March 25, 1922.
3. Rowe, Jeremy, H. Cataract following Thyroidectomy. *British Journal of Ophthalmology*, July, 1919.
4. Committee on Glass-Workers' Cataract. *British Journal of Ophthalmology*, Oct., 1921.
5. Healy, James J. *British Journal of Ophthalmology*, Oct., 1921.
6. Van der Hoeve, J. Schädigungen des Auges durch Licht. *Klinische Monatsblätter für Augenheilkunde*, April-May, 1922.
7. Morax, V. Cataract Operation on Glaucomatous Patients. *American Journal of Ophthalmology*, Aug., 1920.
8. Elliot, R. H. Personal Communication.
9. Fromaget, M. Camille. Cataracte et glaucome: L'extraction de la cataracte est toujours possible dans un oeil glaucomateux normalisé par une operation hypotensive. *Gazette Hebdomadaire des Sciences Médicales*, April 9, 1922.

DISCUSSION ON DR. WEEKS' AND DR. WHEELER'S PAPERS.

DR. G. E. DE SCHWEINITZ, Philadelphia, Pa.: Glaucoma after cataract extraction, a subject always of noteworthy interest, has recently received, among others, special attention from Meller in Austria, Morax in France and Stieren in our country, and as Dr. Weeks has covered the ground with characteristic thoroughness and excellence, it seems advisable to make this small contribution one of personal experience, rather than an attempt to introduce a discussion from the general standpoint.

Among 816 cataract operations, selected from a considerably larger number because the notes are fairly accurate and postoperative observations reasonably prolonged, this complication is recorded thirteen times. Others doubtless have occurred without coming to my attention or knowledge, because it is not an uncommon experience to encounter glaucoma, usually at late periods after cataract extraction, in any large public service in patients who have elsewhere submitted to operation.

As to the type of operation: Nine cases, uncomplicated senile cataract; combined extraction, i. e., with iridectomy and capsulotomy; one case of complicated senile cataract in sense of small cornea, and, as later shown, cupping of discs, combined extraction; one case complicated cataract following syphilitic iridocyclitis, extraction after preliminary iridectomy two cases, extraction of senile cataract after preliminary

iridectomy; one case traumatic cataract, extraction with iridectomy; no cases after simple extraction, i. e. without iridectomy, but these comprise only a small number of the whole series, less than a hundred.

No notable general disturbance in any of the cases, except exophthalmic goitre, one case, severe (malignant?) malaria antedating cataract formation, one case; constitutional syphilis, one case; and indications of myxedema one case, the cataracts being typically of the so-called "black" variety; and constitutional syphilis, one case. All the patients were of senior age, i. e. over fifty-five, except in the case of the traumatic cataract (a boy of about 14), and the cataract following severe malaria (a man of 39). Some of these patients naturally had other signs of advancing years, that is, vague muscular or small joint rheumatism, stiffening of the peripheral arteries, occasional hyalin casts, etc.

None of the records contain any reference to a discovery of increased intraocular tension prior to the primary operation, but all of them except one were noted before the introduction of the tonometer as a practical instrument.

Meller, discussing determining etiologic factors in this postoperative complication, refers to those cases for which no cause can be found; those which are due to iritis and iridocyclitis, followed by adhesions of the iris to the capsule of the lens (posterior synechia); those which follow incarceration of the iris, or of a piece of capsule in the wound; those which depend upon adhesion of the iris to the cornea (anterior synechia); and those associated with retained cortex. In the presence of iritis and its consequences, or iris and capsule incarceration, or retained cortex, the glaucoma is definitely of the secondary type, as, indeed, it is when the activating cause is less definitely demonstrable.

Cases of glaucoma after cataract extraction may be grouped into those which appear early, a few days after operation, and those which arise at a later period, months or even years after the operative procedure. In the present series, the onset of glaucoma as to time was as follows:

Three days after extraction, one case; five days after extraction, one case; one week after extraction, two cases; two weeks after extraction, one case; six weeks after extraction, two cases (one exactly 48 days, the other approximately, exact days of onset not known); eight weeks after extraction (one exactly 54 days), two cases; six months after extraction, one case; one year (approximately) after extraction, one case; three years after extraction, one case; eight years after extraction, one case.

Therefore there are three groups: (a) Early manifestation, i. e. within two weeks, 4 cases; (b) delayed manifestations, that is, from six to eight weeks, 4 cases; long delayed manifestations, i. e. from six months to eight years, 5 cases.

The determining causes were: Incarceration of a delicate thread of capsule in outer angle of wound, 1 case; attack beginning on fifth day, 1 case; incarceration of a tongue shaped piece of capsule in the outer angle of the wound, 1 case, attack beginning about six weeks after extraction, but six days after restoration of anterior chamber, which was unrestored for 42 days; incarceration of iris without iritis, i. e. edge of pillar of coloboma at angle, 2 cases, onset six weeks and three years

respectively after operation; incarceration of iris and slight iritis, 6 cases, onset varying from one week to a number of months (1 case a traumatic cataract 8 years, but there had also been several dissections); retained cortex, 1 case, onset one week after operation; no cause, at least of the character of those described, ascertained, 2 cases, onset three days and more than a year respectively.

Miotic treatment was successful in eight cases; in four of them, this success lasted for years, and may be regarded as permanently satisfactory. In one, rise of tension reappeared at the end of six months and persisted in spite of miotics; operation was positively declined, and the eye subsequently lost completely its visions. In another, the patient disappeared from personal observation, and the history after about eight months is unknown. I more than suspect the ultimate result was not good. Miotics and paracentesis of the cornea utterly failed in one case; further operation was declined. One case came only after glaucoma was well advanced; miotics relieved pain, but that was all; operation was positively declined, and the eye "went bad." Iridectomy, that is freeing the incarceration at the angle, was entirely successful in the cases in which it was performed (two in number); corneoscleral trephining was equally successful (one case).

In this relation of personal experience in glaucoma after cataract extraction, temporary rises of tension, witnessed now and then shortly after or immediately following closure of the wound, that is, restoration of the anterior chamber, have not been included. The manifestations are slight; very faint haze of the cornea, a perceptible "stiffening" of the sclera, etc., easily overlooked. Perhaps the eyes are predisposed to glaucoma, or may respond to an active ciliary hyperemia. The cases are somewhat analogous to rise of tension noted after closure of a corneal fistula, or of an incised wound of the cornea, or of an imperceptible rent of the corneoscleral wall at the periphery of the anterior chamber following a blow, as long ago pointed out by H. Knapp. They are important because if, as is usually the case, a mydriatic is being used, it must be discontinued and a miotic employed; if so, the elevated tension quickly subsides, otherwise a real glaucomatous outbreak may ensue.

Cases of postoperative glaucoma encountered in dispensary practice, as before noted, have also not been included; they have not presented features differing from those described, and were almost invariably seen at a later period.

Cases of mild glaucoma occurring soon after extraction, in which escape of fluid vitreous has been a complication, are also omitted, such, for example, as Meller described, except to mention two cases of this character which have been noted within the past two years. They must be remembered, however, as the continuance of a mydriatic is likely to prove disastrous.

Personal experiences in extraction or expression of cataract in the capsule, according to methods now in vogue, are far too limited in number to be utilized in this discussion. But in two cases seen recently in consultation (both Smith operations), glaucoma was an evident feature. In one, miotics appeared to be reasonably effective; in the other, they had failed, and the eye was beyond the help of any procedure, surgical

or otherwise. They probably were both due to a hernia of the vitreous into the anterior chamber.

While miotics can permanently control the rise of tension, especially that of the mild and temporary type, usually an operation is required. Incarceration of the iris, or capsule, or both, in the angle certainly indicates its excision by iridectomy. Even though the majority of such incarcerations do not cause glaucoma, each one should be regarded as potential in this respect. An eye in which there has been delayed restoration of the anterior chamber should be kept under observation for long periods of time; it may, as has been noted, be subject to an attack within a day or two after the chamber has reformed, but such an attack may be delayed to a much later period. Paracentesis of the cornea seems to be a rather useless procedure, certainly not to be compared with anterior sclerotomy or cyclodialysis, which procedures Meller recommends when the coloboma is normal or the pupil round. Posterior sclerotomy I have not tried, nor have I any faith that it would be effectual. I saw it practiced in one case by a surgeon after a paracentesis had failed, without valuable effect, and in another case it was followed by a severe anterior chamber hemorrhage, and was useless. I have not performed cyclodialysis to control glaucoma after cataract extraction, but have employed it most successfully in cases of return of tension after Elliot's operation and after the Lagrange procedure, and see no reason to doubt the wisdom of Meller's recommendation. Corneoscleral trephining may be considered, if it can be definitely ascertained that the vitreous and anterior chamber are not in communication; it was conspicuously successful in one case in the present series.

In general terms, if there has been iritis or iridocyclitis, or incarceration of the iris or lens capsule, or both, or retained cortex, the onset of glaucoma is not difficult to explain; if the extraction apparently has been uncomplicated, this explanation is more difficult. In some cases, it would seem a hernia of the vitreous may be implicated. Downgrowth of epithelium through the incision into the anterior chamber in rather rare circumstances is a cause of this condition; especially may this be true if there is slow closure of the wound. Even when the extraction has been apparently uncomplicated, it is possible minute examination may reveal a delicate thread of capsule fastened in the edge of the incision. It must be admitted that in most of the cases, exclusive of those eyes predisposed to glaucoma, a fault in operative technic should be incriminated, or perhaps it may be called the development of a complication.

Concerning Dr. Wheeler's admirable paper on cataract after glaucoma, I am tempted to adopt an immemorial custom of some of those who discuss scientific papers, and say, "I have nothing new to add, but should like to emphasize a few points." His concise account of etiologic factors requires no further comment.

A cataract due to injury of the lens capsule during the operation by the keratome, iris forceps or spatula, is, unhappily, only too frequently an operative fault, and the essayist's criticism of descriptions of iridectomy in these circumstances, with advice that the pupillary border of the iris shall be seized, is a timely one and certainly deserves emphasis. Even Meller is not quite precise, although he recommends grasping the iris

"near" the pupil margin, and the greatest of ophthalmic text books which has been rendered into English in this country is probably liable for Dr. Wheeler's stricture, in that the operator is advised to push the iris forceps, its branches closed, "on up to the border of the pupil," where the blades are expanded and a fold of iris is grasped. Perhaps, however, this is equivalent to Dr. Wheeler's recommendation that the points of the iris forceps shall be at least a millimeter away from the margin of the pupil. Two American text books, in their descriptions of iridectomy, come directly within the scope of Dr. Wheeler's criticism; in one of them, in which the present speaker has a not unnatural interest, this fault has been corrected in the last edition, and he apologizes for previous derelictions. These references are inserted because this matter deserves, as has been said, decided emphasis.

Another factor in producing cataract after operation for glaucoma, is one which resides in useless and dangerous manipulations to expel blood from the anterior chamber. Some of them are almost equivalent to Foerster's method of artificial ripening, that is, triturating the lens fibers by rubbing the cornea over the coloboma with a horn spoon. I entirely agree with Dr. Wheeler's contention, that in cases of cataract associated with glaucoma, the opaque lens should be extracted if light perception remained, in the hope that some visual improvement may result. An interesting experience in this respect is the following: In the case of a man with only one eye, and that containing a complete glaucomatous cataract, and with a light field only on the temporal side, the lens was extracted after preliminary iridectomy, with uncomplicated healing. The disc was deeply cupped and atrophic, but the light perception on the temporal side was converted into object perception, which enabled the man to steer his way around a room without colliding with the chairs, etc., in it. The man lived long after the operation, and a more grateful patient I have never had.

DR. W. H. WILDER, Chicago, Ill.: No subject engages the attention of ophthalmologists more than that of glaucoma, partly for the reason that the condition is one that does not have an analogue in the pathology of any other part of the body, and partly because its pathogenesis is, after all, so little understood. The study of it has aroused no end of speculation and numerous hypotheses as to its real cause; but with due respect to the Weber-Kneiss and other theories, we must confess that we have not solved the problem of the real cause of glaucoma.

The features of the condition that are set forth in the theories of Weber-Kneiss, Priestley Smith and others are more or less mechanical factors that may depend for their operation on something deeper, possibly of a chemical nature, that may influence the quantity and the character of the secretion of the ciliary body, the aqueous humor. I think we must reckon with this and not believe that all cases of glaucoma, even those of secondary type, are originally brought about by the mechanical glueing of the iris to the corneal angle, although this feature may be a conspicuous one.

In the subject under consideration the glaucoma is frequently of the secondary type, and the mechanical factors probably predominate. Dr. Weeks has described these conditions, but it may not be amiss to again emphasize some of them. Remnants of cortex may block the

spaces of Fontana, hence the importance of removing all such at time of operation, with irrigation of the anterior chamber if necessary.

Displacement of the capsule and adhesion of a tag of it to the corneal wound at the time of the cataract extraction or later following the discission of the capsule—such a tag of capsule dragging on the ciliary body—would tend to irritate it and cause congestion, if it did not actually excite an iridocyclitis.

Treacher Collins has demonstrated, that a strand of capsule adherent to the corneal wound after a discission may pull the iris forward, and so help to block the filtration angle. He states that this is more likely to occur if the puncture for the discission is made far forward in the cornea, hence the importance of introducing the discission needle near the limbus and away from any anterior synechia that may have been left after the cataract extraction.

In this connection a word in regard to the treatment of the capsule at the time of its extraction may not be amiss. The capsule should not be divided by criss-cross incisions with the cystotome, for small tags of it may be drawn into the wound and become incarcerated there. One straight vertical incision, or a vertical incision with a short horizontal one added to its upper end like an inverted "L," would seem to be a better technic. Removal of a portion of the anterior capsule with proper forceps is preferred by many operators, and personal experience has convinced me that it is a valuable method.

Downgrowth of epithelium through the corneal wound into the anterior chamber, as demonstrated by Dr. Fuchs, must be very uncommon, but it may occur.

The importance of the proper replacement of any iris entanglement, and also of the conjunctival flap, during the toilet of the cataract wound should be emphasized. Care should be taken that the conjunctival flap is not infolded, especially at the ends of the incision, for this might cause a delayed closure of the wound.

Exudate from a plastic iritis after extraction may cause the iris to adhere to the remains of the capsule, and so interfere with the proper circulation of the aqueous. All these conditions may act as mechanical factors to bring about glaucoma. But in any of these conditions, there may be the added factor of alteration of the character or quantity of the aqueous secretion. This is probably illustrated in the case of a leaky wound following extraction, in which the anterior chamber does not reform for several days, possibly on account of imperfect healing. When the chamber does reform there may be serious rise of tension, and this has been explained on the ground of an alteration of the character of the aqueous, possibly a greater albumin content, so that the spaces of Fontana are blocked.

Iridocyclitis after cataract extraction, when there is apparently no mechanical factor present, would seem to furnish another illustration of glaucoma resulting from altered secretion. Hence the importance of prompt treatment of cases that show too great reaction following extraction. Large doses of salicylat of soda as suggested by Gifford, together with hot applications, are valuable remedies. In some more severe cases, I have recently used protein therapy with gratifying results, giving 50 million dead typhoid bacilli at a dose subcutaneously, repeating it three

or four times if necessary. However, it is comforting to think that glaucoma following cataract extraction is not of frequent occurrence, and my own experience would incline me to agree with the estimate that it does not occur in more than 1 per cent of cases.

As to cataract secondary to glaucoma, so well presented by the paper of Dr. Wheeler, my observation has been that the opacity of the lens begins with a haziness of the nuclear or paranuclear portions, rather than with distinct striae or patches of opacity in the peripheral parts. Such faint central opacities are easily demonstrated with the corneal microscope and slit lamp illumination. But one should not neglect the use of the test with the Purkinje images in such cases. As the image from the posterior capsule is made to approach that from the cornea, it is seen to grow much dimmer or to disappear entirely as it nears the hazy zone of the nuclear portion of the lens. This is an extremely valuable test in certain cases, and is often neglected in a careless examination. As to the cataract that sometimes follows after operation for glaucoma without any injury to the lens or its capsule, may it not be explained by the reduction of the long continued intraocular pressure on the lens, allowing the lens tissue to expand, with possibly separation of the lamella and formation of minute vacuoles?

But one could also suspect that the relief of the increased pressure might alter the character of the secretion from the ciliary body in such a way as to affect the nutrition of the lens. But all of this, in our present knowledge of the cause of glaucoma, is largely speculation.

DR. ALLEN GREENWOOD, Boston, Mass.: There are two important points in Dr. Weeks' remarks, which I wish to note. One is, that in considering glaucoma following cataract extraction, one should rule out those cases with a predisposing condition to glaucoma. I am inclined to believe that, in many cases, surgeons operate for cataract without noting a tendency to glaucoma previous to the time of operation. I think it is very difficult to pick out among cataract cases those that have no predisposition to glaucoma. When one takes into account the fact that many cases of chronic glaucoma with tension below 20 have lately been reported, it is easy to see how it may happen that these cases are overlooked when cupping cannot be observed because of a cataract. Therefore, it is not possible to rule out glaucoma at the time of extraction.

In regard to Dr. Wheeler's remarks as to the position of the incision in extraction of cataract after a well done trephine, I have, personally, preferred to go in front of the trephine opening and try to extract the lens in the capsule. I am glad to say that the two cases I have done in this way have been very successful, leaving a good filtering cicatrix behind the cataract incision.

I am also wondering, in reference to Dr. Wilder's remarks in regard to iridocyclitis, how much, in the future, the elimination of the sensitivity of the eye to lens protein will have to do with the prevention of iridocyclitis.

DR. WALTER R. PARKER, Detroit, Mich.: Since our program came out, I have made an attempt to review some of the records of my cataract cases. I have been able to go over 483 in detail. From these cases have been eliminated all that were suspected of having had glaucoma before operation. In the 483 cases, glaucoma developed after operation

four times — .82 per cent. I did not have a late record of all the cases, consequently the incidence of glaucoma is not necessarily accurate. In one of the four cases that developed glaucoma, the lens was extracted in the capsule by the traction method. In three cases, the combined operation with capsulectomy was performed. In all the cases, the iris pillars were more or less adherent to the wound. In no case was there an actual prolapse. In one case, the pillar of the iris was freed, followed by pilocarpin and general treatment. After several months' time, the tension was normal, and the patient has had no subsequent trouble, although the eyes have been under observation several years. In another case, pilocarpin failed to reduce the tension. The eyeball was painless, and the patient refused operation, retaining a good looking, blind eye. In each of the other two cases, a trephine operation was performed at the site of the coloboma. In both cases, the operation failed to reduce the tension. In one, the wound closed, and the patient retained a painless, blind eye; and in the other case, at the time of the operation, the patient had pain, loss of vitreous and all the symptoms characteristic of profound vitreous hemorrhage. The eye was afterwards enucleated.

My experience is very limited in trephining at the site of the coloboma for relief of glaucoma, after the lens has been removed. The impression in these few cases, however, has been so profound that I hesitate to recommend it as a routine procedure.

DR. GEORGE A. MOORE, Palmer, Mass.: I have wondered, sometimes, when, after doing an operation for the relief of glaucoma, a cataract developed, if I had unwittingly contributed to its cause. I came to the conclusion in later cases, when very great care was exercised, that the operation was not responsible. Also, in the past five or six years, I have encountered a few cases which seemed to be indubitably glaucoma after (and because of) cataract. By cataract, I mean lens changes very likely to escape observation behind the iris, especially in cases where one avoids a mydriatic.

This leaves out of the reckoning those not infrequently found cases where a disturbed lens is palpably the cause of a frank glaucoma. I do not include the so-called overgrown lens, intumescent or traumatic cataracts, but incipient cataracts which, having their beginning *before*, are not discovered until *after* increased tension has brought the patient for advice. May it not be that in many cases photochemical activity is then exerted upon an already altered lens, as in incipient cataract?

DR. EDMOND E. BLAAUW, Buffalo, N. Y.: I think we can go a step farther and say that we can diagnose a cyst in the anterior chamber. Here is a section of the cornea, anterior chamber and iris diaphragm. As you move the light from one side to the other, you are absolutely sure it will show the slightest inequality of the anterior chamber. Next, if there is any membrane you can detect it right away. I think that is a practical point that may be of some little value in favor of the slit lamp.

DR. ARTHUR J. BEDELL, Albany, N. Y.: I ask your attention to two things: I do not think a previous coloboma has anything to do with the production of a cataract following a glaucoma operation. If this were so, we should see in the clear colobomatous area the earliest cataractous changes. We do not. In the production of cataract following glaucoma

operation, there are certain things that can be seen. At the margin of the coloboma, the iris is bound by a mass of exudate to the capsule, and surrounding this exudate there is a definite capsular and subcapsular opacity. This is increased by the traction of the iris on the mass. In the pupillary area, there are subcapsular striae and, at times, opacities in the anterior cortical layers. The posterior cortical type, with its branched outline, is the same as with other intraocular changes, such as extensive choroiditis.

DR. JOHN E. WEEKS, New York City (closing): There are just one or two points I wish to speak of. One is the probable change in intraocular fluids, making these fluids less readily diffusible and changing the condition of the aqueous, so that it does not escape through the lymph spaces as readily as normal. In my opinion, such a change takes place not infrequently. In cases of iridocyclitis, we must have such a change. In all probability, where particles of cortex are left we will have that change, and in case hemorrhage takes place from the retina. It seems to me that research in that direction gives promise of very large improvement in our knowledge of the cause of glaucoma, particularly in this class of cases.

Another point is the ingrowth of epithelium. In my search through the literature, I have failed to find any case where ingrowth of epithelium took place in a wound not located in the cornea.

Dr. Greenwood spoke about ruling out preexisting glaucoma. That, we must all admit, is a very difficult thing to do. But if we are careful in taking our tension, even by palpation, and wherever there is a suspicion of anything above normal taking it with a tonometer, we can come very close to telling whether the case is one of hypertension before extraction.

DR. JOHN M. WHEELER, New York City (closing): In regard to Dr. Moore's question, I might say that there are many cases in which it is not possible to say with absolute certainty which was the incipient trouble, glaucoma or cataract; but neither Dr. Weeks nor I referred to that in our papers.

In regard to Dr. Bedell's question, the presumption is not correct. It is not known how light affects the lens and causes degeneration, but it is known that degeneration does not occur especially in the pupils when it is produced experimentally. Van der Hoeve has dealt with that, and says that in all probability the cataract is developed by scattered rays of light, and it is possible that the effect is primarily in the ciliary body and not the lens; at any rate in experimental cataract, the lens opacity does not develop in the exposed areas particularly.

THE EYE IN EXPRESSION AND REPRESENTATION. NOTES ON OCULAR PHYSIOGNOMY IN ART AND LIFE.

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Like so many other departments of human thought, physiognomy has been the battlefield of science and superstition. Astrology and the mythology which linked the qualities of human beings with attributes of their patron gods, or still more mystically with a guiding star, are instances. The pseudoscientific correlation of metals with planets also had its reverberations in physiognomy, as indicated by such designations as Saturnine, Mercurial, Martial, Jovial, and the like.

Evolution, Darwin's observations and biologic conclusions shed light on the origins of facial gesture, and established the essential unity of human and animal expression or physiognomy.

With increased knowledge has come, *pari passu*, a less contemptuous attitude toward the ideas of olden times and even those of primitive man. We have found out that fact is not only stranger than fiction, but often enough stronger than theory, and that it will not do to deny a thing because we cannot explain it. The phrase, "it stands to reason," covers a multitude of philosophic sins both of commission and omission.

The approach to physiognomy from the medical standpoint is manifold. The most obvious is the recognition of the fact that certain well defined clinical complexes have their foreshadowings in facies and function within the limits of health or at least of well-being. Another factor is that endocrine dominance, which undoubtedly produces specific states of immunity, reaction to disease and drugs, and characteristic qualities, also produces specific morphologic and structural changes. To infer the one from the presence of the other, is justified both logically and empirically.

Not unlike other branches of medical science, that of Physiognomy has been characterized by its contemptuous attitude toward the standpoint of yesterday and its cock-sureness about the standpoint of today. It is almost amusing to read, e.g. in Mauthner, pitying references to the unscientific and mystic trend of thought in the Physiognomes of antiquity, who attributed definite animal characteristics to humans who showed facial peculiarities of the

ox, the sheep, the goat, the rabbit, and so on. There is, after all, not much that is new under the sun, and in these days of endocrinology, we might not be quite so ready to condemn utterly the empiric, if not the scientific, basis of this view. Certainly, the studies of Crile, Cannon and others have shown that animals, as well as mankind, have their generic endocrine makeup as well as special peculiarities. The nervous horse, the placid ox, the timid, nervous, tachycardic, tremulous hare, have, also, a physiognomy, and it is by no means absurd to infer, or at least to accept as a possibility, that an endocrine dominance which produces the skull form or the face and eye form peculiar to the ox will also tend to produce a bovine expression in humans. On the other hand, it is our turn now to pooh-pooh and deride the teleologic or aprioristic point of view, which explained all facial expression as the result of habitual gesture, and all gesture as the indication of a purposeful action. Thus, the glaring eye with its wide open lid fissure and prominent globe was accepted as the natural, nay, the inevitable expression of an intention to see as much as possible of the object of ire, victim or antagonist. The fact that the same wide eyed factors are present in extreme fear or horror, as also in ecstasy or wonder without marked emotional coloring either aggressive or fugitive, indicates sufficiently that such a generalization, while not, perhaps, devoid of all basis of fact, is far from complete and satisfactory. A study of Physiognomy, today, cannot fail to be influenced by two lines of thought, both of which are of comparatively recent development. The study of the glands of internal secretion has progressed to a point where all but the most bigoted must admit the important influence they exert on morphology and function. Skull form, face form and, so, eye form will depend, at least to a degree, on structure as determined by endocrine balance.

The glance, the rate of eye and of lid motion, the habitual fixation mode, convergence mean, and individual horopter, what we might call the "ocular stance," are, also, largely matters of temperament or habitual endocrine dominance, as much if not as strikingly in health as in disease.

On the other hand, expression, as we employ and understand the term, implies an observer, at times we may say a partner, and as this observer is, generally, a human being, his interpretation of the eye and its glance became a matter of experience, and then of tradition, and, today, also of symbolism. The meaning depended not only on the original function or purpose indicated by the eye glance of the moment, but on ideas associated with it or in

the course of time attached to it, sometimes quite arbitrarily. The glance was, *ab initio*, an important means of communicating information, the more valuable in that it could convey a warning or a threat silently and thus confidentially. Much of the present significance of eye gesture such as the wink, the ogle, or the scowl, may trace back to such primitive acts.

So the eye gradually took on the role of an organ of speech, and the language of the eyes uses many of the figures of rhetoric and of imaginative literature. Psychoanalysis shows us that many of the processes which take place in the dream are found also in primitive speech, fable, and folk form. Symbolism, condensation, transfer, paradox, representation by contraries or opposites are all mechanisms common to these apparently independent fields of human thought. If we wish to understand a man's thought, whether voluntary or involuntary as hidden, repressed or sublimated desire, we must take these things into consideration, and thus the judgment of disposition or character takes on a new meaning and acquires methods of study and instruments of precision from a comparatively new science. There is nothing essentially devout about raising the visual axes above the horizontal, but this significance has become more or less artificially attached to that movement of the eyes since mankind placed its God in a heaven. Nor is there anything, *per se*, of modesty in the downward gaze, unless it takes into consideration the onlooker, the *vis-à-vis*, friendly or antagonistic, and realizes the expression of a fear to meet the gaze; a desire, real or feigned, to escape from love or battle.

Many of the commonest expressive gestures or positions are associated with original purposive function; thus the laugh, a baring of the teeth in anticipation of a savory mouthful. The original function has been supplanted or supplemented by others, but the gesture remains as man's most instinctive expression of pleasure, joy, and thus as a derived component of physiognomy. This derivative character also applies in many ways to the intellectual or spiritual connotations. The incredibly complex facial play takes much of its meaning and its inner suggestiveness from associations so submerged, instinctive, and unrecognized, that while we may, and generally do, get the full meaning of a glance however subtle, we find it most difficult to analyse or describe either the physiognomic factor itself or the emotion or train of thought which it has enabled us to sense so fully.

The riddle of personality has always troubled the mind of man. It was tacitly assumed that expression was an index of

disposition, character, and, to a certain degree, of habitual reaction to physical and psychic stimuli. It was soon recognized that expression, facial or other, is the result of repeated gesture, resulting in a functional stereotype, or in one which is morphologic, anatomic, or physiognomic. The judgment of man on his fellows was closely bound up with his ideas of the world in general, and tribal views of heaven and earth are largely indicated by their attribution of dispositions and character. Fable and folk lore, as well as language, bear witness to this fact. It holds good to-day as in the childhood of the race, for we shall see that comparatively modern theories, such as evolution, and the biologic viewpoint explain many things which were formerly enveloped in the mists of metaphysics and symbolistic mysticism. The medicine man, the witch-doctor, the healer, the priest, and later the physician, all realized that there was a difference in the reaction of individuals to noxious as well as beneficent agents, to the influences of nature as well as to the healing effects of drugs, and sensed a possibility that a clue might be given to the individual reaction in his physical appearance or in his habitual expression and function. The last two are factors which merge one into the other. Respiration and rate of circulation, gait, carriage, rhythm of speech, are instances of this, having to do with physiology as well as with morphology and structure.

Physiognomy is not a simple manifestation or series of manifestations, but an interpretation, and as in all such reciprocal reactions between human beings, we must bear in mind not only individual reactions, but such things as tradition, prejudice, suggestion, repressions and much else that has become intelligible to us since the advent of Freud and his analyses of the subconscious mind.

To take merely one example, a certain expression of the eyes is taken to denote candor and simplicity, and is spoken of as an innocent look. Why a wide eyed somewhat fixed regard, with slightly divergent visual axes or parallelism, should denote an absence of guile does not at once appear. It was explained, not so long ago, on very mechanical-physiologic grounds, as indicating a willingness to see and be seen, hence an easy conscience. The speech of the people, often right without knowing why, calls this gaze the "baby stare," and thus at once indicates why it is supposed to stand for innocence. The expression of religious devotion with eyes upraised to the heavens is another instance of literary symbolism accepted as biologic fact. There would be nothing particularly devout or religious in the raising of the

visual axes above the horizontal were it not that mankind places its God or its gods up there, and the glance in that direction has acquired a meaning of its own which has really nothing to do with physiognomy or expression, as such, but everything to do with traditional dogma.

More and more we are forced to realize that the general form and external appearance of the human body depend, to a large extent, upon the functioning during the early developmental period of the endocrine glands. Our stature, the kind of faces we have, the length of our arms and legs, the quantity and location of our fat, the amount and distribution of hair on our bodies, the tonicity of our muscles, the sound of our voice, and the size of the larynx, the emotion to which our exterior gives expression—all are to a certain extent conditioned by the productivity of our glands of internal secretion.

Ocular expression is largely a question of adnexa, brows, eyebrows, lid fissure, and interpretation of the same is largely traditional, suggestive, and transferred or symbolic.

Paradigms of normal physiognomy are found in the Pantheon, on Olympus; those of morbid physiognomy in the clinic. As each of the Hellenic gods had his or her prime characteristic, so their representations in sculpture conveyed to the people the artist's idea of facial expression most expressive of that character. The face of Mars is the face of courage, the face of Venus that of ideal sexual charm, that of Jove the incarnation of power, majesty, and benevolence.

We recognize in many a facies of disease merely an exaggeration of types which are seen about us in daily life.

The observation of the race, preserved in phrase and fable, was accurate, however incorrect their philosophy or their logic.

It is fair to assume, that some at least of the factors, which in excess produce typical disease pictures, are responsible for tendencies within the bounds of normal physiognomy, or on the border line, where the expression is similar.

A STUDY OF THE BACTERIOLOGY OF THE NORMAL AND INFLAMED CONJUNCTIVA WITH SPECIAL REFERENCE TO THE PRESENCE OF THE STREPTOCOCCUS AND PNEUMOCOCCUS.

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MONTREAL, CANADA.

The bacteriology of the conjunctiva, both in health and disease, is a subject of interest and importance to ophthalmologists. A knowledge of the organisms of the diseased conjunctiva is important, because of its relation to diagnosis, treatment and prognosis. A knowledge of the bacteria of the normal conjunctiva is of importance, because of its relation to ophthalmic surgery. The conjunctiva, with its moist surface, continuously bathed with lacrimal fluid, closely associated with the nasal mucous membrane, constantly exposed to infections in various ways, offers every inducement to the lodgement of microorganisms. It seems, indeed, one of those parts of the body which form a bacteriologic home—ready, easily accessible. It has been established that numerous harmful as well as harmless bacteria are frequently present in the normal conjunctiva. The researches of Sattler, Fick, Michael, Weeks, Koch, Morax, and Axenfeld, have clearly established that fact.

Gallenga studied the normal conjunctiva, and reported his findings to the Italian Ophthalmological Congress in Genoa in 1886. Later reports came from Petresko, Leber, Gaubert, Gifford, and Sattler. During the winter of 1902-03, at the instigation of Dr. W. G. M. Byers, the writer undertook the examination of a series of normal cases. Of the 140 examined, 40 were negative. The organisms isolated in the 100 positive cases were as follows:

Staphylococcus Pyogenes Albus	48 times
Staphylococcus Epidermidis Albus	9 times
Staphylococcus Pyogenes Aureus	twice
Streptococcus Pyogenes	16 times
Bacillus Xerosis	42 times
Bacillus of Diphtheria group	once
Sarcina Lutea	once

As will be seen from this table, the staphylococcus, streptococcus, and bacillus xerosis were frequently present. Since that

period, I have been constantly interested in the bacteriology of both the normal and inflamed conjunctiva. My results have corresponded with those of many others, in that frequently pathogenic microorganisms were isolated from normal conjunctivae; that different forms of staphylococci and streptococci were sometimes present, so that we must always consider the possibility of the presence of such harmful organisms as the streptococcus, pneumococcus, Morax-Axenfeld diplobacillus, colon bacillus, and others.

Any one interested in this line of work, however, must have been struck with the comparatively large number of negative results. This matter has interested me for years, for in both healthy and diseased conditions of the conjunctiva, I have felt the percentage was far too large, and have constantly been looking for methods which might give more accurate results.

The following apparatus in one's consulting room or clinic will enable one to do thorough bacteriologic work. For the examination of the smear, one needs, of course, a platinum wire and glass slides. The preparation of the slide is very important, and many negative results are due to lack of attention to this detail. A small stand containing four or five small bottles, for gentian violet, Gram's iodine, absolute alcohol, and safranin will enable one to do Gram's stain right there. One should have for culture purposes a few tubes of either bouillon or salt solution. These can be readily inoculated, and sent to the hospital for routine examination.

While considering a change in my technic of routine examination, there was published the work of James Harvard Brown on "The Use of Blood Agar for the Study of Streptococci," (The Rockefeller Institute for Medical Research, 1919). This consists in a detailed study of the appearances produced by the growth of streptococci in blood agar. Three types of growth are described.

TYPE ALPHA. After 48 hours incubation, the change produced by streptococci of this type may be described as a somewhat greenish discoloration and partial hemolysis of the blood corpuscles immediately surrounding the colony, forming a rather indefinitely bounded zone, 1 to 2 millimeters in diameter, and surrounded by a second narrow, clearer, not discolored, partly hemolyzed zone. The inner, discolored, zones were fairly constant in size and composition on all the plates.

TYPE BETA. Streptococci of this type produced hemolyzed zones on horse blood agar plates radically different from those

of the Alpha type, in that they were sharply defined, clear, transparent, completely hemolyzed, colorless zones, 2 to 4 millimeters in diameter.

There was nothing particularly distinctive about the surface colonies of either type on blood agar plates. All were round, glistening, translucent, flatly convex colonies, surrounded by hemolyzed greyish zones, corresponding to their respective types.

TYPE GAMMA. By the Gamma type is meant the growth of the streptococcus colonies within and on the blood agar plate, without the production of any hemolysis or discoloration of the surrounding medium during incubation or refrigeration.

These three types in blood agar plates are fairly distinctive, so that on seeing a streptococcus colony for the first time in a blood agar plate, there is rarely any difficulty in deciding to which type it belongs. Incubation for 24 to 48 hours, followed by refrigeration, is usually sufficient to determine the type. Insofar as Brown has been able to observe, not only do all the green producing and brown producing streptococci belong to the type Alpha, but also the pneumococcus and streptococcus mucosus (pneumococcus). There was a general tendency for both green producing streptococci and pneumococci to produce less hemolysis and more greenish discoloration of corpuscles in human blood agar than in horse or rabbit blood agar.

With this article as a guide, the examination of a series of normal and inflamed conjunctivae was undertaken. The following technic was carried out:

Where it was possible to obtain material enough to stain, a smear preparation was made and stained with Gram's method. Many of these give negative results, because in many cases of normal conditions it is naturally difficult to get material to stain. On the other hand, it is surprising what numbers of bacteria one sometimes finds on one slide.

In normal cases, the upper lid is then gently rubbed over the eyeball to stimulate the flow of tears, and then the lacrimal fluid is collected in a sterile pipette from the inner canthus. Small tubes, which contain one cubic centimeter of sterile salt solution, are now inoculated, and this material is later taken to the laboratory. Tubes of bouillon and blood serum are now inoculated, and blood agar plates are made in the usual manner.

The whole is now incubated for 24 hours, studied, and re-incubated for 24 hours; studied, and then the plates are put on ice for 24 hours, and again examined. The plates are examined for hemolysis after 24, 36, and 48 hours incubation. Hemolysis

is best shown, as a rule, after 48 hours incubation and 24 hours in the refrigerator. As soon as a growth appears, single colonies are picked off and studied by

1. Hanging drop—for morphology.
2. Gram's stain.
3. Neisser's differential stain for diphtheria.

Where diphtheroids are found, single colonies are transplanted on blood serum slants and incubated for further study of

1. Morphology.
2. Staining reaction.
3. Biochemic reactions on sugar media.

A series of examinations were made by this method, and perhaps one or two cited cases will suffice to illustrate our results. J. A., with senile cataract of right eye, after the usual simpler examination with negative results, was operated upon by me with loss of the eye from panophthalmitis. The left eye was as free from congestion as the right had been. With a sterile pipette some lacrimal fluid was collected, diluted in bouillon, and blood agar plates made. Examination after incubation showed small, deep, hemolytic and nonhemolytic colonies in a fairly profuse growth. The hemolytic colonies, when examined, were found to be Gram plus cocci in chains. This was later classified as streptococcus subacidus. The nonhemolytic colonies were the bacillus xerosis. There is not the slightest doubt in my mind that the right eye was lost through the action of streptococci, the presence of which would have been shown, had one known enough to have used improved methods.

I have always thought that extirpation of the inflamed tear sac left the chance of infection remote. A. L. had his lacrimal sac removed by me some years ago, before the war. He happened along while we were examining cases, so some of his lacrimal fluid was collected, bouillon inoculated, and blood agar plates made. A profuse growth of pneumococcus was obtained.

Mrs. B., a refraction case, with normal conjunctivae, gave a profuse growth, with the colonies quite green after twenty-four hours in the incubator—pneumococcus, with a few colonies of staphylococcus epidermidis albus. Mrs. A., with slightly hyperemic conjunctivae, gave a negative smear. The blood agar plate gave a profuse growth of the streptococcus nonhemolyticus.

These results speak for themselves. I do not wish to be an alarmist, and am glad to say that we have had many negative results to balance against these. Nevertheless, enough cases have been cited to show that, with improved technic, our knowl-

edge of the bacteriology of the normal conjunctiva may be considerably improved.

On the inflamed conjunctiva, the streptococcus and pneumococcus play important roles. The former is a very definite factor in those severe membranous processes, mostly seen in poorly nourished children, which cause severe necrosis and run a virulent course. Luckily, these cases are rare. The etiologic factor is very easily demonstrated in both smear and culture. In the Montreal area, the pneumococcus is by far the most common cause of acute conjunctivitis, and is seen as frequently in adults as in children. I have never noticed here any special predisposition for children. As a rule, the pneumococci are found in enormous numbers during the progress of the disease to its height. They then rapidly disappear. This has been noted by Axenfeld. One should not be surprised, therefore, when examining a case in the receding stage, if pneumococci are not found. Koch-Weeks' conjunctivitis is a great rarity in certain districts. I have seen very few cases of it in Montreal, although last Summer I did see a family of children so infected. The clinical feature which impressed me was the excessive amount of discharge. The bacilli, very fine slender rods, are usually found in large numbers in the leucocytes, free, and in clusters. They resemble the bacillus of mouse septicemia, and are not unlike bacillus influenzae, though longer and thinner. Morax-Axenfeld conjunctivitis is described as a chronic catarrhal inflammation. I have only one observation to make about it, and that is, that it may be as acute as either the Koch-Weeks' or pneumococcus variety. This will be best illustrated by the lantern slide to be shown. The pus from gonorrheal ophthalmia often returns a negative report. Two slides to be shown were made from such cases. The method employed was described by me some years ago, and has been used constantly ever since. Without question gonococci will be demonstrated by this method from pus where the old way would leave one with "report negative."

CONCLUSIONS.

1. Normal conjunctivae may harbor pathogenic organisms without symptoms.
2. No major surgical operation should be performed upon the eye without first carefully examining the lacrimal fluid for pathogenic organisms.
3. For the thorough examination of the normal conjunctiva, in any case, cultures are necessary.
4. The examination by smear alone gives many negative

results in cases where pathogenic organisms will be easily demonstrated by further bacteriologic methods.

5. Pathogenic microorganisms, such as streptococci and pneumococci, are best demonstrated by the use of blood agar plates. This method is simple, and the presence of either streptococcus or pneumococcus may be easily demonstrated in 24 to 48 hours.

I wish to thank Dr. L. J. Rhea, director of the Department of Pathology at the Montreal General Hospital, who assisted me in many ways, also to acknowledge that Dr. Trossman was associated with me in part of this investigation.

DISCUSSION.

DR. WILLIAM C. FINNOFF, Denver, Colo.: Dr. McKee has given the subject of bacteriology of the eye a great deal of thought. His present paper gives us his conclusions after twenty years of careful study of smears and cultures from the conjunctiva. I am glad that the importance of careful bacteriologic examination of our cases of conjunctivitis has been emphasized. I also wish to indorse his statement, that smears can be made easily in any office. The equipment that is required is inexpensive, and the time that is necessary for the examination is slight. There are only a few bacteria that are pathogenic to the conjunctiva, and any practicing ophthalmologist should be able to make smears and recognize the organism after examination with the microscope.

For several years, I have made smears in all of my cases of conjunctivitis. At least 85% of the acute cases were due to the pneumococcus. In my private and clinical practice, I have never found a smear that contained Koch-Weeks bacilli. Ours is one of the localities in which this form of conjunctivitis is rare.

In 1911, the essayist published a paper on the histopathology of diplobacillary conjunctivitis, in which he pointed out that the diplobacillus was found on the surface and between the epithelial cells of the conjunctiva. I had hoped that his present paper would elaborate this theory.

One of the reasons that smears taken from the conjunctiva are so frequently negative, is because most pathogenic bacteria are epithelial parasites, and are confined to the epithelial cells in the early stage of the inflammation, and smears of the secretion do not contain organisms.

It is true that we frequently find bacteria in the conjunctival secretions when there are no signs of inflammation. This, however, is to be expected, because the conjunctiva is constantly exposed to air and dust. Pneumococci are often found, but are frequently saprophytes, and only become pathogenic when the globe is opened or perforated and a favorable soil supplied for their transformation from saprophytic to pathogenic bacteria.

In 1921, Lindner published in the Fuchs Festschrift a comprehensive article on the nature of pathogenic bacteria in the conjunctiva.

He pointed out that the majority of the organisms are epithelial parasites. In the beginning of a conjunctival inflammation, the bacteria are few in number and are present in small clusters or plaques on the surface of the epithelium. At this stage, it is impossible to find them in smears which contain conjunctival epithelium, unless the area on which the plaque is located is removed with the applicator or loop. As the colony increases in size, the organisms invade the adjacent epithelial cells, and at the same time are transplanted to other portions of the bulbar and palpebral conjunctiva by the movement of the lids. As the bacteria increase in number, their toxins, through their chemotactic action, call forth serum and later pus from the subepithelial blood vessels. The serum separates the epithelial cells, and the bacteria pass between them and invade the deeper layers, as well as the protoplasm of the cell. Later, the epithelium is desquamated to a varying degree, and the serum and pus escape and form the secretion that is present in an acute conjunctivitis. The bacteria are washed from the epithelial cells into the secretion, and their presence in the latter is secondary. Therefore, in addition to the methods suggested by the author, attention should be directed toward the epithelium. Smears of the epithelial covering of both the bulbar and palpebral conjunctiva can be easily obtained after cocaineization, by rubbing away the superficial layers of epithelium with a firm platinum loop or preferably a platinum spatula. The material is then placed on a cover glass or a glass slide, and fixed and stained in the ordinary manner.

I had the good fortune to spend some time with Lindner in his laboratory a year ago, and obtained lantern slides of his splendid photomicrographs, a few of which I will have thrown on the screen.

DR. HARRY S. GRADLE, Chicago, Ill.: It seems that we must differentiate rather sharply between the normal conjunctiva and the bacteriology of the inflamed conjunctiva. The two present entirely different problems. As regards the former, I believe the first careful work was that of Elschmig and Ulbrich, published in 1909, in which they examined the conjunctivae in cataract cases before operation, and reached the most astonishing conclusion that 43 per cent of normal appearing conjunctivae in patients who came for cataract extraction, contained pneumococci and streptococci. The best results were obtained by the use of horse serum and bouillon mixed, one part horse serum and two parts bouillon. A few drops were put into the conjunctiva, and the fluid then aspirated in a pipette and incubated for 24 to 48 hours, after which growth became manifest. At the same time, I examined a series of 100 cases in the Elschmig clinic, where cultures were made. Before the cultures were made, we made the usual smear with a platinum loop. In 50 per cent of cases that showed pneumococci and streptococci by culture, we were not able to show bacteria by the smear method alone. In other words, in normal conjunctivae there are apparently organisms that are not brought to view by ordinary methods of smear. Therefore, when we attempt to solve the bacteriology of the normal conjunctival sac, we must resort to the methods used by Elschmig and Ulbrich and simplified by Dr. McKee. On the other hand, if we are dealing with an inflamed conjunctivae, it is essential that we search for the epithelial parasites which Dr. Finnoff has mentioned.

The beautiful work that Lindner published is merely carrying it a step beyond the work published by Dr. McKee in 1904, in which he showed the nature of the epithelial parasites causing conjunctival disease. A few years later, Brown Pusey published an article showing the nature and character of the Morax-Axenfeld bacillus.

In the examination of conjunctivae for organisms causing disease, cultural methods are not always necessary unless we care to go deeper into the nature of the organisms. As a rule, an epithelial smear will suffice, and we have found that this can be made without any annoyance to the patient by gently stroking the conjunctiva with a small flat spatula. It is not sufficient to be satisfied with the examination of the secretion alone, as we do not arrive at the true nature of the causative organism.

DR. WILLIAM L. BENEDICT, Rochester, Minn.: Bacteriologic examination of the conjunctivae, if it is to be of any particular value, must take into account other things besides cultural methods and methods of examination that are matters of laboratory technic. We know that organisms have a variation in their virulence; that in certain atmospheres or certain climates they will vary in their ability to produce disease, and that in their resistance to the defense that is built up in the body organs, they are harmless at one time of the year, and in the same individual become extremely dangerous at other times of the year.

Also, our cultural methods, particularly for differentiating the types of streptococci and pneumococci, are not sufficient unless we take into account all of the different conditions under which the organisms will grow and will maintain their virulency. Organisms that are developed in the presence of air, such as Dr. McKee has spoken of, tend to lose their virulency in their second or third generation of propagation, but when they can be grown under proper conditions, will retain their marked virulency. If we take pus from an infected area and grow it in long tubes of broth, so we can have development of different organisms in different layers of the broth, we will find that certain types will grow at the bottom, others at the middle, and others at the top. These organisms, taken out separately and injected into animals, will have different degrees of virulency. Anaerobic organisms grown at the bottom of the tube, where the oxygen concentration is lowest, will retain their virulency, but when we take these same organisms and develop them on agar plates, they will be perfectly innocuous.

Another point we must remember is the seasonal variation in organisms. We know that we are subject to common colds and infections of the nasal mucous membrane at all seasons of the year, but our waves of influenza and pneumonia which have the highest virulency and produce the greatest fatalities occur in certain seasons of the year, particularly in November and December.

The organisms that are really producing the serious menace to health are those preponderant only during certain seasons of the year, and our ordinary cultural methods give us very little practical evidence of their virulency, while some organisms can be differentiated by cultural methods.

EYE CONDITIONS OBSERVED IN CERTAIN TYPES OF THE FEEBLE MINDED.

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In the United States, the word "feeble minded" includes all grades of so-called "congenital" mental defect, from the lowest to the highest type. Our observations of the eyes of this class of patients were conducted at the Polk State School, an institution for the feeble minded, at Polk, Pennsylvania. This institution cares for 2,108 of these unfortunates. In our investigations, a sufficient number were selected for study from groups which showed characteristics distinct enough to classify them into types; and the eye conditions were as carefully noted as possible under the circumstances.

It might seem that our observations were not so complete at times as the cases demanded, but we would remind you that the mental defective does not respond like the normal individual, and in many cases it requires the utmost patience and tact to make an eye examination which is satisfactory in any degree. There are but few if any conditions of the eye which are alone peculiar to the feeble minded and may not be found in the normal individual; but when we note the development of the eye as being from the anterior cerebral vesicle, and that the retina is really a prolongation of the brain substance, it is reasonable to suppose that ocular defects are of more frequent occurrence among the feeble minded than among normal people.

Gilbert states, however, that the eye may frequently escape the influence of morbid causes which affect the brain, because it is fully formed at an early period of fetal life. Blindness from birth or early infancy may occur along with mental defect; or when it occurs in conjunction with defect of some of the other senses, the mind may be so impoverished as to bring about a condition of true mental defect—the "amentia of deprivation." The growing brain cells must be stimulated by vibrations transmitted through the special sense pathways. This is shown by the

poor development of the occipital cortex that occurs in congenital blindness, which fact has been utilized in mapping out the visual area in the brain. Sensations are not only thus necessary for growth; they are also material out of which thoughts and ideas are built, the sum total of which constitutes mind. Should,



Low grade Mongolian imbecile.

Theresa H. Age 8 years. Born, Pennsylvania. Austro-Hungarian parentage. Long difficult labor—60 hours. Began to walk at two years of age. Mental peculiarity noticed by parents at three years of age. Last born of a family of four children. Mother 42 years of age at time of birth. Low grade Mongolian imbecile.

therefore, a single sensory avenue be blocked, the mind must forever remain the poorer by the impressions which should have entered through this channel.

The most clearly defined of all classes of mental defectives is that of mongolism.* The most striking physical characteristics of this type are that the physiognomy shows a resemblance to

*See picture of Mongolian imbecile of a low grade.

the Mongolian race, with a brachycephalic head and narrow slit like palpebral fissures, converging downward and inward. The face is broad; the nose small, broad and saddle bridged, with nostrils frequently directed forward. The ears are small and project outward. The tongue shows characteristic deep transverse fissures. Eczematous areas are frequent around the mouth. The hand is broad, fingers short, and the little one nearly always curves inward. The mental grade varies from slight enfeeble-



Low grade Mongolian imbecile. Aged 10 years.

Edison H. Age 10 years. Low grade Mongolian imbecile. Mental peculiarity first noticed at two years of age. Began to walk at three, and talk at four years of age. Ninth born child. One born after. Followed by five miscarriages.

ment down to the idiot. The majority have a mental age of about four years. The mongol is usually good natured and affectionate. This type is said to be congenital but not hereditary.

Contrary to the statement of most authorities, the Polk Institution has cared for cases of mongolism in which other members of the family were mental defectives. Of eleven boys and fifteen girls of varying degrees of mental defect, which were chosen from a group of forty cases of the mongolian type, the following were the eye findings:

Of the twenty-six cases, three had blepharitis squamosa, complicated with styas and a mild type of chronic catarrhal conjunctivitis. Five had blepharitis ulcerosa with severe chronic catarrhal conjunctivitis, trichiasis and hypertrophy of the lid margins. Four of these showed pronounced ectropion. Blepharitis eczematosa occurred in five cases, and was associated with eczema about the mouth and slight ectropion. In thirteen blepharitis cases, there were five of corneal opacity, which varied in intensity from nebula to leucoma. Numerous dotted opacities were seen in the lenses of four subjects when the pupils were fully dilated. Vision did not seem to be affected from the presence of the opacities. The presence of double lamellar cataracts was noted in two children who were rachitic subjects, as shown by the irregular and imperfect development of the teeth. The opacities were not very dense, hence fair vision was retained. Epicanthus was seen in nine cases, which increased their Mongolian like expression. The condition of epicanthus was complicated with convergent strabismus in three individuals. One subject had complete cataract in one eye, with iritic adhesions. Bilateral nystagmus of the horizontal type was present in two leucomatous cornea subjects. The fundi in two cases of disseminated choroiditis showed spots of pigment surrounded by a pale margin and crossed by retinal vessels. The patches were located principally in the periphery of the fundus. Nine had anomalies of pigment in the iris. Seventeen had normal fundi. The youngest member of the group was four years of age, while the oldest was twenty-two.

Of the different types of feeble mindedness, none is more strikingly characteristic than microcephalus,† which may be traced through a series of gradations, from idiocy and imbecility to simple "feeble mindedness." In these cases, the head is noticeably small, narrow, with rapidly receding forehead, a somewhat pointed vertex and flat occiput. The frontal and parietal lobes of the brain are small, but the most striking arrest in development is seen in the occipital and temporosphenoidal lobes.

"The theories that this is an atavistic variation or the result of premature synostosis have been disproved; the small skull is simply the envelope of the brain of which the normal development has been arrested, probably about the fifth month of gestation."¹

†Microcephalic idiot.

Dr. Tredgold states that "microcephalics usually come of a pronounced neuropathic stock; their brothers and sisters are often typical degenerates, and frequently one or more of them suffer from the same condition." This is exemplified in the following cases.



Microcephalic idiot, 12 years of age.

(1) Antonia M., born in Pennsylvania, and of Italian parents; she is eleven years of age and an inmate of the Polk Institution. This girl is a microcephalic idiot, and is subject to epileptic fits. A brother and sister are similarly affected. She can neither walk nor talk, but hears well, and has teeth of the Hutchinson type. The left eye shows slight divergent strabismus. The cornea and media are clear in both eyes. The

grey color and sharp outline of the optic disc of the left eye presents a typical picture of simple atrophy. The temporal side of the right optic disc is decidedly grey, while the nasal side is normal in color. The surrounding fundus and vessels of both eyes are healthy. The fields and vision could not be taken



Antonia M. Age 11 years. Born Pennsylvania. Italian parentage. Microcephalic idiot. Epileptic. Paraplegic. A brother and sister in similar condition.

owing to the low mentality. The ophthalmoscopic pictures in this case are similar to that seen in multiple sclerosis.

(2) Harriet H., aged twenty years, a microcephalic idiot and sufficiently intelligent to do certain work when supervised; small of stature; gait shuffling. Three members of her family were similarly affected. Cornea and media normal. Motility of the eyes unimpaired. Right optic disc very white, outline

sharp, lamina markings obscured, all vessels of the fundus narrowed. Apparently this might be classed as neuritic optic atrophy. The left disc is reddish grey; the veins somewhat tortuous.

(3) Fanny K., aged twelve, microcephalic idiot, born in the United States and of Austrian parents, both of whom are living and healthy. Another child is an epileptic, walks with a shuffling gait and speaks a few words when prompted. She is small of stature, has notched incisors, slight lateral nystagmus and sluggish pupils. The ophthalmoscopic picture is similar in both eyes, in that the media is clear, the macular region has a dirty



Hydrocephalus.

yellowish cast with dust like pigment, which is also seen throughout the retina but in less profusion. The retinal vessels are narrowed and slightly tortuous. The optic disc of each eye is distinctly white on the temporal side. The nasal sides are normal. The mental condition precluded taking the vision or fields. This case might be classed as one of maculo-cerebral degeneration.

(4) Harry T., microcephalic idiot, aged nineteen, was delivered by use of forceps. Mother and father healthy and intelligent. Walked well at three years of age. Later walked with a shuffling gait. Became apathetic and degenerated into the present type. Tremor and nystagmus absent. Pupils react. Media clear. Bilateral simple atrophy of the temporal halves of the discs. Nasal sides normal. Remainder of each fundus normal. Vision does not seem to be greatly impaired.

The four cases which have been described in detail were chosen because they showed rare ophthalmoscopic pictures. A number of others of a low grade type of microcephalus were examined ophthalmoscopically, and several were found with optic atrophy in a variety of stages, but they were not sufficiently typical to warrant special mention.

In hydrocephalus, the mental defect varies greatly and is not always proportionate to the deformity. The ocular symptoms of this disease result from pressure on the optic chiasm by distention of the third ventricle. Two cases will illustrate the eye condition usually found.

(1) Martha Y., bedridden from birth. Cannot talk, but smiles and emits guttural sounds. Head exceedingly large. Media clear. Nystagmus. Double optic atrophy, not complete. Fundus vessels tortuous.

(2) Ralph C., aged twelve. Jewish boy. No family history. Low grade imbecile. Can talk some. Walks fairly well. Head quite large. Media clear. Fundus best seen with a —5.00 lens. Vessels tortuous. Has beginning double atrophy.

In the absence of, or congenital dysfunction of the thyroid, we have a condition known as Cretinism, with its marked retardation of mental and physical development.

The imperfect physical development of this affection is well illustrated in the group of four cretins which are shown in the picture. Harry, No. 1, and Cornell S., No. 2, aged thirty-eight and thirty-six respectively, are brothers, born of American parents. Harry is a low grade imbecile, while Cornell is classed as an idiot. Both have enlarged thyroids. Annie H., No. 3, aged thirty-nine years, and Sarah H., No. 4, aged thirty years, are not related. They are both low grade imbeciles, and were born in Pennsylvania. Family history unknown. The most common ocular signs and symptoms seen in the cretin are as follows: There is considerable width between the eyes, which are deep in the orbit, and seem small from the pseudoedema of the eyelids. The interpalpebral fissure is narrow and horizontal. The superciliary hair is scanty. The pupils, media and fundi are normal in most cases. (See p. 208, 209.)

Acromegaly (Marie's disease) is an affection regarding which many theories have been advanced, but the etiology and pathology remain obscure. The most striking single change found in most cases on postmortem was the enormous enlargement of the pituitary gland. The following case well illustrates the condition



- Cretins. (1) Harry, age 38 years, and (2) Cornell, age 36 years, are brothers. (3) Annie, age 39, and (4) Sarah, age 30, are not related.
- No. 1. Harry S. Age 38 years. Born, Pennsylvania. American parentage. Cretin. Low grade imbecile. Thyroid moderately enlarged.
- No. 2. Cornell S. Age 36 years. Idiot. Cretin. Thyroid enlarged. Brother of No. 1.
- No. 3. Annie H. Age 39 years. Born, Pennsylvania. Cretin. Low grade imbecile. Condition recognized by parents at one year of age.
- No. 4. Sarah H. Age 30 years. Born, Pennsylvania. Cretin. Low grade imbecile. Family history unknown.



Cretins, 16 and 18 years old.

- No. 1. Ethel I. Age 18 years. Born, New York City. Austro-Hungarian parentage. Cretin. Thyroid enlargement. Family history unknown.
- No. 2. Sara I. Age 16 years. Born, New York City. Sister of No. 1. Cretin. Condition recognized by parents at three years of age. Probably congenital. Was placed on thyroid treatment at age of 16 years and made very decided improvement. Talks a great deal more than she did. Takes an interest in things about her, apathy has disappeared, and spontaneous, reflected actions are carried out. Instead of a torpid manner there is gay and sprightly conduct. The mucoid condition and distended abdomen have disappeared and the soft parts have become quite normal. She has lost 12 pounds in weight, and digestion has improved. Also, sexual development and establishment of the menstrual function.



Acromegaly.



Acromegaly.

of acromegaly: William T., a middle grade type, age thirty-five years; six feet four inches in height; weight 180 pounds; born of American parents, both of whom are intelligent and healthy. William was a normal child until seven years of age, when he contracted a severe illness, the nature of which is not known to the parents. After this sickness, he commenced to develop the present disfiguring overgrowth of the hands, feet and jaws, together with slight weakness, drowsiness and failure of vision. An X-ray examination showed enlargement of the pituitary gland. Eyes normal as far as could be determined by a visual and ophthalmoscopic examination. Bitemporal hemianopsia, slight on the left, and more pronounced on the right side. Vision of the right eye 20/200; of the left eye 20/100. Under a cycloplegic and by use of a retinoscope, the vision of the right eye was corrected to 20/60 with a +2.00 cylinder axis 90, while the left eye could read 20/50 with a +1.25 cylinder axis 85.

An interesting case of double congenital coloboma of the iris and choroid was seen in a middle grade type of feeble minded boy, fifteen years of age, whose family history could not be obtained. The iris tissue in the left eye was absent from the inferotemporal part of the iris, extending from the pupillary border to the ciliary border, which gave the characteristic keyhole coloboma of the iris. In the right eye, the coloboma was in the form of a narrow slit, which widened out slightly at the ciliary margin, and allowed a glimpse of the front part of a coloboma of the choroid beneath an anterior capsular cataract, which was present in this eye. The coloboma of the choroid of the left eye extended almost to the disc, and was marked by an oval glistening white area, with an irregularly pigmented border. Several retinal vessels crossed this space. A number of sclerotic patches were present throughout the fundus of this eye. The lens did not share in the coloboma.

Three brothers, who were classed as the "middle grade" type, were affected with double congenital cataracts of the anterior capsular variety. Their ages were respectively twenty-eight, thirty-four and thirty-six years. No definite family history could be obtained. Two of them were operated upon in childhood, with the result that fair vision was obtained. The third one was not operated upon, as the cataracts were incomplete. The cornea in each case was perfectly clear. A brother and sister belonging to another family were similarly affected.

Errors of refraction occur in a large percentage of cases, hyperopia being the most common deviation. Of two hundred

cases examined here, eighty per cent were found to be hyperopic. Myopia is of less frequent occurrence, being found in nine per cent of the cases. Astigmatism is also frequently found. The range of variation was from one-half to ten diopters. The cases selected were ones in which visual defects were known or were supposed to exist, consequently the percentage is higher than would be found were the cases taken in order.

These cases are of special interest on account of the improvement noted in many of them after being properly corrected, although all cases are not amenable to correction. Glasses among the low grades, of course, are impractical for obvious reasons. However, in a number of apparent low grades, the proper fitting of glasses, enabling the child to see, has practically promoted him to a higher grade. In all grades, the improvement in vision must be marked, or the relief of eyestrain quite appreciable, or they do not care to wear the glasses after the novelty of them is over. But in those cases which are indicated, such improvement will be noted in the child's application and progress as, compared with his previous record, leaves unquestionable the value of this means of helping the child. The common method of testing the vision by means of Snellen's test charts is of limited use, being applicable only to those cases which can read. The shadow test, by means of the retinoscope, is the most practical, and can be used in all cases where cornea and media are clear. It is accurate and obviates the necessity of relying upon the statements of a child. The average eye, however, especially in hyperopia, will not stand full correction, but if a child can read, the test types will be valuable aid in determining the amount of deduction to be made. Among the motor disorders we find nystagmus and strabismus, the latter being quite common.

The appended visual tests will give an idea of the refractive errors in some of the medium and high grade types of feeble mindedness.

Joseph W.

Retinoscope	{	O.D.	+3.50	sph.	⊖	+2.50	cyl. axis	90
		O.S.	+4.50	sph.	⊖	+ .50	cyl. axis	120
Subjective	{	O.D.	+3.00	sph.	⊖	+1.50	cyl. axis	90 = 6/9
		O.S.	+4.00	sph.	⊖	+ .50	cyl. axis	120 = 6/30

Opal P.

Retinoscope	{	O.D.	+5.50	sph.	⊖	+1.25	cyl. axis	90
		O.S.	+5.50	sph.	⊖	+1.00	cyl. axis	150
Subjective	{	O.S.	+4.00	sph.	⊖	+1.00	cyl. axis	90 = 6/6
		O.D.	+4.00	sph.	⊖	+1.00	cyl. axis	165 = 6/20

Lucille R.

Retinoscope	{	O.D.	+2.50	sph.	⊖	+1.50	cyl. axis	45	
		O.S.	+2.25	sph.	⊖	+1.50	cyl. axis	135	
Subjective	{	O.D.	+1.50	sph.	⊖	+1.50	cyl. axis	45	= 6/9
		O.S.	+1.50	sph.	⊖	+1.50	cyl. axis	135	= 6/6

Joseph J.

Retinoscope	{	O.D.	+4.00	cyl.		axis	165	
		O.S.	-3.50	cyl.		axis	165	
Subjective	{	O.D.	+3.25	cyl.		axis	165	= 6/15
		O.S.	-2.75	cyl.		axis	165	= 6/12

Thomas B.

Retinoscope	{	O.D.	-7.00	sph.	⊖	-3.00	cyl. axis	90	= 20/40
		O.S.	-7.00	sph.	⊖	-3.00	cyl. axis	90	= 20/40

Lizzie C.

Retinoscope	{	O.D.	-6.50	sph.	⊖	-2.00	cyl. axis	90	= 6/30
		O.S.	-6.00	sph.	⊖	-2.00	cyl. axis	90	= 6/30

BIBLIOGRAPHY.

1. Shuttleworth and Potts. Mentally Deficient Children.

DISCUSSION.

DR. EDWARD STIEREN, Pittsburgh, Pa.: Having preceded Doctor Jobson by about fifteen years as visiting ophthalmologist to Polk, I appreciate the difficulties under which he has labored in making these examinations, gathering the statistics and tabulating them. Polk is an ideally managed institution—a large happy family, where the high grade children are taught the three R's in school each day, the older ones are taught the various arts and crafts, and everything is conducted as nearly as possible like normal life. The eye defects of course are not found so much in high grade imbeciles as in the low grade, and for that reason examination is much more difficult.

Some of these inmates are intensely interesting. One low grade microcephalic has only one redeeming feature, that is, he is absolutely reliable in delivering mail. That was his job, to deliver mail throughout the institution, and nobody could get a letter that was not intended for him. He was quite a low grade type, and on several occasions we attempted to refract him and succeeded in getting a mydriatic in his eyes, but as soon as we threw the light into his eyes, he would fight and scream like an outraged animal and we never did learn what the error was.

Then there was another boy, who was hyperopic in one eye and myopic in the other. He was refracted and glasses were given him, but in a week he reported he had lost them. Another pair was bought, but he lost them, and then they found he was trading his glasses for pets. His cornet was taken from him, so that he could not play in the band, and he was told that when he found his last pair of glasses his cornet would be returned. He lost no more.

One of the highest degrees of hyperopia I have ever seen was at Polk, 24D. hypermetropia in each eye. Another case was a boy who had a genius for mathematics. He could add as fast as you could

give him six or eight numbers, four figures each, and he would give you the correct answer every time. He could not read print when it was erect, but read everything upside down. This boy apparently had no mental defect except that he would set things on fire—arson was his obsession.

It is painstaking work, and I think Doctor Jobson deserves great credit for gathering and tabulating these defects, and giving them to us in the admirable form of his presentation.

DR. B. A. BLACK, Polk, Pa. (closing): The types of cases selected in this report show a great deal more pathology of the eye, as well as general pathology, than the average feeble minded.

Reference was made in the paper to a series of 200 cases examined, where ocular disturbance was known or supposed to exist. In another series of 200 cases, from the moron and imbecile group, taken consecutively as classified in the cottages, and examined with special reference to refraction, 84 per cent were found with the various forms of hypermetropia as follows: 37.5 per cent, simple hyperopia; 4.5 per cent with simple hyperopic astigmatism, and 42 per cent with compound hyperopic astigmatism. In 1 per cent of cases, mixed astigmatism was found, and in 12 per cent the various forms of myopia.

With regard to the pituitary cases, there seems to be a special liability of the optic nerve to be involved, whether from an extension of the hypertrophic struma beyond the confines of the sella, or due to a primary infundibular growth. Consequently, the degree of implication of the chiasm, nerves or tract bears no direct relation to the size of the sella.

The atrophy in these cases is the so-called primary atrophy, and we find no edema of the disc in the early stages. However, as the condition progresses until the growth has reached such a size as to lead to general pressure phenomena, due in a vast majority of cases to an occlusion of the foramen of Monroe and a resulting hydrops of the lateral ventricles, we may have a choked disc superimposed on the atrophic nervehead.

I think we are safe in saying that the amblyopia associated with a primary atrophy is due more to a physiologic bloc to light impulses than to actual destruction of the nerve, because in many cases where the pressure is relieved by operation, a fair degree of vision has been re-established.

Perimetric deviations are demonstrable in a majority of cases. However, the supposedly typical bitemporal hemianopsia with a vertical meridian bisecting the macula is not always found. In fact, homonymous hemianopsia may occur in a number of instances. In three cases reported by Cushing, which came to autopsy, and where the source of the lateral pressure was disclosed, it was found that the glandular struma had burst beyond the capsule and extended upward along the left side of the chiasm in two of them, producing a right homonymous defect; and along the right side of the chiasm in the other, producing a left homonymous defect.

I think we should also emphasize the fact that it is rare to find equal involvement of the two eyes in these pituitary cases.

RESECTION OF THE OPTIC NERVE.

L. WEBSTER FOX, M.D., LL.D.

PHILADELPHIA, PA.

The term "resection of the optic nerve" has been used so long in connection with a certain ophthalmic procedure, that it has become fixed and definitely indicates only that operation, although it actually describes a different surgical procedure that is seldom performed. The operation, as most commonly em-



No. 1.

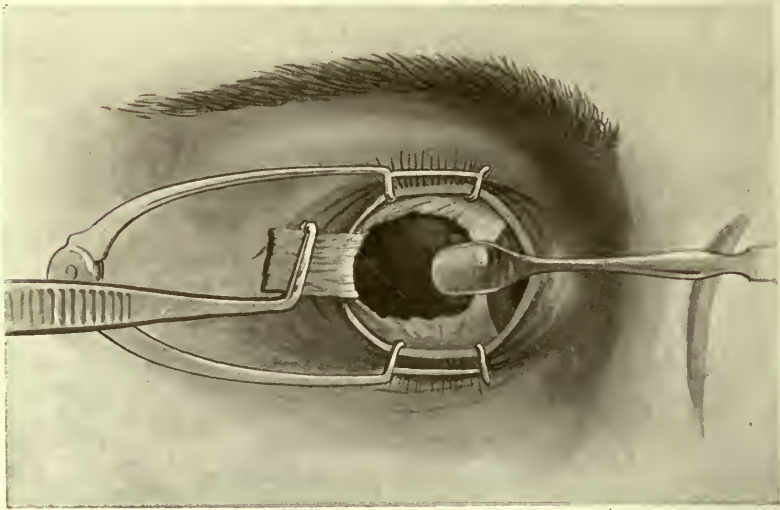
Fig. 1. Resection of the Optic Nerve performed on the left eye of the rabbit. Photograph was taken six weeks after the operation.

ployed, consists of the severance of the optic nerve from the eyeball; the other operation, that of removing a portion of the nerve, being so seldom performed, this faulty nomenclature must be retained.

There is not a very great field of application for this operation, but when it is indicated, it is a most satisfactory therapeutic measure, in that it meets its indication promptly and effectively. Until the problem of glaucoma has been effectively met, there will be encountered a definite proportion of cases in which failure to control the disease, and worse than all, failure to control the most prominent symptom, to wit, pain, has followed all medical

and surgical treatment. In these cases, we have left enucleation. The removal of the painful organ is followed by cessation of the pain. While the morbid physiology of glaucoma is still a matter of speculation, this one feature is definite and an absolute fact—removal of the painful, diseased globe relieves the pain. But in the case of amputation elsewhere in the body, this is not always so. Not infrequently there is so-called “referred” pain—pain experienced by the patient in members that are no longer there.

Now enucleation has many great disadvantages, especially to



No. 2.

Fig. 2. Clearance of eyeball prior to cutting the optic nerve.

the man or woman not in the lower walks of life. Argue as one may, an empty orbit or one occupied by a glass eye is a handicap, socially and professionally. Literature, when it desires to create or depict a villain, has him one eyed or decorated with a black patch over one eye. Thus, the impression of the connection between one eyed individuals and villainy of the deepest dye has become universal, and to meet it various measures have been taken to retain the natural globe in the orbit.

Prominent business and professional men are not infrequently the subject of glaucoma and diseases of the cornea, and with the luck that follows such instances, will take to themselves the worst forms of the disease—the fulminating, the malignant and the progressively painful types—and resisting, as they do in many



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.

instances, the orthodox treatments, go from bad to worse, until conservation of the vision becomes of secondary consideration to the relief of the pain. In such cases we think of enucleation, but must be chary of its employment. Therefore, we turn to severance of the nerve from the globe, and while we do not completely sever all the structures in the orbit from the globe, when we perform this operation, we do relieve the pain, even though the accepted view of the pathology of the disease does not credit the optic nerve with the production of pain.

The operation employed by Pagenstecher, Rohnur, de Wecker, Meyer and others, included the actual removal of a portion of the nerve, and was advocated for sympathetic ophthalmia, but even its most enthusiastic supporters relegated it to second place when compared with enucleation.

The operation I have employed in painful, blind glaucomatous eyes, or eyes blinded from other complications, is performed essentially as follows:

Under général anesthesia, the lids are separated and maintained so by the ordinary spring eye speculum, and to make the opening still wider an external canthotomy is performed. A vertical incision is then made through the conjunctiva over the insertion of the external rectus muscle, if the left eye is the one to be operated upon. The conjunctiva is then dissected off as far back as the external canthus will permit. This serves to expose the muscle completely. If the right eye, the incision is made over the internal muscle. The operator faces the patient in either case. Two silk threads are then passed through the muscle near its tendinous insertion. The muscle is afterwards divided and drawn to the temporal side, thus exposing the globe beneath it. All the overlying tissues should be separated from the eyeball by curved scissors. A long strabismus hook (Fig. 7-C), bent at a right angle, is then inserted along the globe until the nerve is found, after which it is brought forward by means of the hook (Fig. 7-D). A duck bill retractor (Fig. 7-B) is then passed downward until the nerve is encountered, when it is pressed down and out, keeping the adjacent tissues out of the way. A second bent hook is also passed backward and inserted under the optic nerve. A portion of the nerve is then exposed and severed by the large de Wecker compressing scissors (Fig. 7-A), especially devised for this operation, which controls the hemorrhage that follows cutting the central artery. The eyeball is then rotated forward and a small portion of the bulbar end of the nerve is excised by means of scissors. The eyeball should be rotated into its proper place

and the external rectus adapted to its normal position on the eyeball by means of the thread previously described. The conjunctival wound is closed by sutures which may be removed in three days. While there is, as a rule, much less reaction than would be expected, it is well to apply antiphlogistic dressings* for several days.

This description covers the operation I have performed over

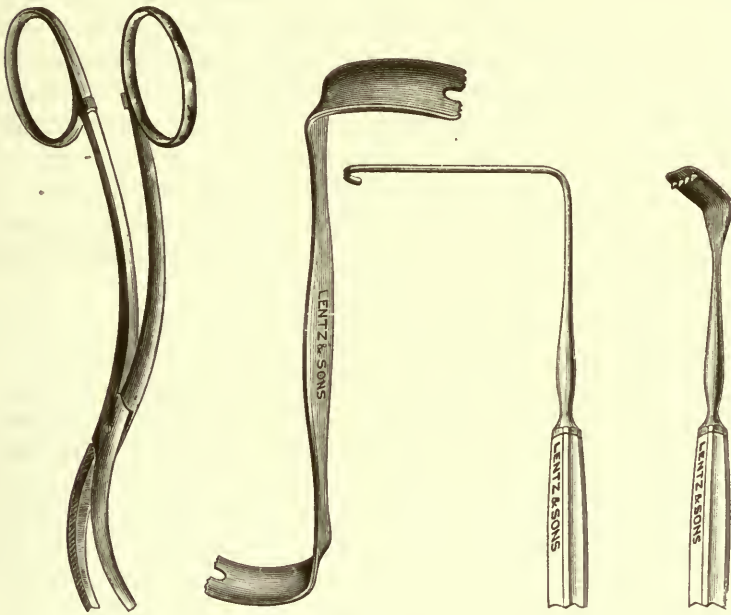


Fig. 7. Instruments used in resection of optic nerve.

- A. De Wecker's Scissors. B. Fox's Duck Bill Retractor. C. Right Angled Strabismus Hook. D. Carter's Retractor.

a period of many years, but increasing familiarity with the structures within the orbit, and their behaviour under the stress of surgical traumatism has led me to discard some of this operative technic in the more recent cases. Thus, I have found that external canthotomy is not always necessary, as a wide exposure

*Lotion.

EXTERNAL LOTION

Liquor plumbi subacetatis dil.....	3 ii
Tincturæ opii	
Tincturæ belladonnæ	aa..... 3 iss
Tincturæ arnicæ	3 i
Aquæ camphoræ	
Aquæ destillatæ	aa q. s. ad.... 3 iv
M. ft.	
Sig.—Apply locally as directed.	

of the globe with extreme rotation to the opposite side will often bring the nerve within the field of operation, even though not within the field of vision of the operator. Again, and what is more important, the excision of a portion of the nerve is not absolutely necessary, hence the statement in the early part of this paper regarding the nomenclature. It may so happen, in the wide exposure of Tenon's capsule, we are also severing microscopic structures that are factors of great importance in the production of the pain, of which we are at present in sublime ignorance.

In most cases, there is little or no trophic disturbance of the eyeball, and when it does occur, it is questionable if this is related to the operation, or is due rather to the advanced trophic changes incidental to the progressing glaucoma. In one case of advanced painful glaucoma, where the operation was performed upon a physician, the shrinking that followed was very nicely disguised by his wearing a high plus spherical lens, which magnified the globe to those that looked directly at him.

If in rare instances the de Wecker scissors fails to clamp the posterior end of the nerve after a severance has been made, we are likely to have a profuse hemorrhage, saturating the post-orbital tissues and producing a marked proptosis. Extreme pressure with the surgeon's hand against the closed eyeball will stop the bleeding. The external muscle is then stitched in place, and a compress bandage applied. This is not removed for three or four days, at the end of which time the closed lids are bathed with warm sterile water and another bandage applied, and this is kept moist with an antiphlogistic lotion. (See p. 219). The stitches are allowed to remain and remove themselves, or they may be taken out at the end of six or seven days. Reduction of swelling goes on rapidly.

My early acceptance of the operation was greatly influenced by the work of de Wecker, although the previous work of Meyer, Schweigger, Pagenstecher and Pflüger had failed to impress me with its merits. The early operations were followed by proptosis, postocular hemorrhage, and phthisis bulbi. It was due to de Wecker's technic and instruments that these were eliminated.

While the operation was originally devised as a substitute for enucleation in sympathetic ophthalmia, there has been such a variance in the results of different operators, and often unreliability in the experience of any single surgeon, that it is best to reserve its use in this affection for the selected case.

In eyes blind or nearly so from glaucoma, or other diseases that might recur as the result of the presence of that particular eye, it affords a most excellent means of rendering that organ inert, while at the same time retaining the comestic feature of a human eye in a human orbit.

Before performing this operation on the human, a number of operations were performed on rabbits, and in no instance was the detached nerve near the severed end of the eyeball when the globe resumed its natural position. The drawing attached illuminates the text. The photographs show the patients upon whom the operation was performed during the past year. This operation is applicable to defective eyes beyond any hope of restoring vision.

DISCUSSION.

DR. WILLIAM L. BENEDICT, Rochester, Minn.: In recent years, there has been a revival of consideration of substitutes for enucleation of the eye. First, a small number of persons who are advised to sacrifice an eye prefer to keep the globe if it is not unsightly, rather than wear an artificial eye. Second, the physical condition of the person, particularly if advanced in years, may make it desirable to do some minor operation to relieve the condition for which help was sought, with less shock than accompanies an enucleation. Third, following the removal of cysts of the orbit, tumors of the optic nerves or other tissues in the posterior part of the orbit, so much contraction of the remaining tissues takes place that an artificial eye cannot be fitted, while if the eye can be left in place, its bed can be built up by substitution of tissue, so that a satisfactory position of the eye is maintained, often with good motion.

The number of cases requiring a substitute for enucleation is relatively small, and aside from the wishes of the patient, (and there are few who object to enucleation when it is indicated) is kept down by the larger factor of safety to the fellow eye that is afforded by enucleation.

Implantation of a foreign substance, such as cartilage or glass balls, into Tenon's capsule following enucleation gives, in most instances, a very satisfactory cosmetic result, with greater safety than any substitute operation can afford. Implantation of substances into the scleral capsule following evisceration gives, as a rule, no better cosmetic results than implantation into Tenon's capsule, and the reaction following the operation of evisceration is often prolonged, and does not afford the protection against sympathetic ophthalmia that is obtained by enucleation.

The greatest need for substitute operations for enucleation is found in cases of painful glaucoma in aged persons, and in cases of orbital growth that does not involve the globe, when the eye can be saved by a Kronlein operation, or by a method that approaches the growth without interfering greatly with the musculature within the orbit. For such cases, opticociliary neurotomy is a satisfactory

procedure, and may be safely employed. It should not be employed, however, if there has been advanced degeneration of the uveal tract, or in cases where the absence of ocular tumor or massive hemorrhage within the globe cannot be demonstrated. Eyes that have been blind and painful for years from uveitis with secondary glaucoma, are not suitable to save and should be enucleated. The technic of optico-ciliary neurotomy will vary with the operator, and with the condition found in the orbit at the time of operation. The earlier advocates of this operation approached the nerve from the nasal side, but I believe that greater working space is usually found on the temporal side. If possible, the internal rectus should be left intact, for the fine adjustment for position necessary to a good cosmetic result depends upon integrity of the ocular muscles. Hemorrhage into the orbit is to be avoided by some such device as Dr. Fox has mentioned, but if it occurs, a drain should be inserted. Marked edema of the orbital structures and lids often follow this operation and is difficult to reduce. It may cause corneal sloughing from pressure and make enucleation necessary. Unless the posterior half of the globe is stripped, some ciliary nerves are likely to remain uncut, and the eye continues to be painful. One patient on whom an optico-ciliary neurotomy was done, continued to have pain in the orbit even after the eye was enucleated at a subsequent operation.

DR. S. LEWIS ZIEGLER, Philadelphia, Pa.: I began performing optico-ciliary neurotomy about thirty years ago, after visiting De Wecker's clinic and witnessing his method. I have found it very satisfactory in every respect except one. Division of the external rectus is objectionable from a cosmetic standpoint. If you have a large post-operative hemorrhage, the globe will be pushed forward so far that the muscle sutures will be torn out, and leave you with an internal squint. I therefore modified my technic by omitting the tenotomy and following the technic of Boucheron. I also modified De Wecker's clamp scissors by making them straighter. This allows you to lean the scissors farther over and secure a larger section of the optic nerve. It makes no difference, however, whether you simply divide the nerve or resect a piece of it, as there will always be enough hemorrhage to keep the ends apart. In spite of the scissors clamp on the optic nerve, you are likely to have a secondary hemorrhage, which I have seen leak out and spread through the cellular tissue of the face. Your only protection is a tight pressure bandage kept on for at least two days.

The operation of neurotomy of the optic nerve was first suggested by Arlt in 1853, and later endorsed by von Graefe. Rondeau, in 1866, began the additional section of the ciliary nerves, and proposed the formal designation of "optico-ciliary neurotomy." Boucheron, in 1876, first attempted the operation without section of the recti muscles. He opened the conjunctiva between the superior and external recti muscles, and pulled the eyeball far forward to facilitate division of the optic and ciliary nerves. This was the technic adopted by Knapp. Schoeler, in 1875, began to make a section of the external rectus, in order to gain a larger operative field. Schweigger revised this technic by cutting the internal rectus, first, however, suturing

or clamping the muscle so that it would not escape from him. Pagenstecher began practising this technic about 1884, but was not a strong supporter of it as was De Wecker.

The two things to be guarded against are hemorrhagic proptosis and relaxation of the cut recti muscles, if you perform tenotomy. Optico-ciliary neurotomy preserves a good looking eye, with excellent movement, and free from sympathetic danger.

DR. JOHN S. KIRKENDALL, Ithaca, N. Y.: I had the pleasure of caring for a Mrs. Champaign, whom the doctor operated upon about a year since. When first seen by me, there were a few nodule of granulations at the point of attachment of the muscles that had been divided. About a week later, I found a stitch which I removed, following which all was quiet, tension was normal, with a very satisfactory cosmetic effect, which has continued up to the present time.

DR. L. WEBSTER FOX, Philadelphia, Pa. (closing): I am pleased to have brought this operation to the attention of the members here, because I feel it has a field, and when carefully and properly performed, I think will be beneficial to many of your patients.

A CASE OF EMBOLISM OF THE INFERIOR DIVISION OF THE CENTRAL RETINAL ARTERY WITH LATE RECOVERY OF CENTRAL VISION.

E. TERRY SMITH, M.D.

HARTFORD, CONN.

While partial retinal embolism is a sufficiently rare condition to be of interest, the symptoms and progress in the present case are so unusual as to deserve special consideration.

The patient is a female, 31 years of age. Occupation, telephone operator.

On August 3, 1922, while at work, the patient noticed sudden loss of vision in the upper field of the left eye. Careful inquiry elicited no prodromal symptoms.

When examined by Dr. Colman W. Cutler, of New York, during the latter part of September, 1922, the findings were as follows: V. O. D. 6/6, V. O. S. no fixation. The entire upper part of the left visual field, including the fixation point, was lost. The nerve presented pallor at its inferior and temporal quadrants suggestive of atrophy. The lower temporal branch of the central retinal artery was almost obliterated, and, in general, the other arteries were smaller than usual. The retina showed remains of edema, downward and outward, and the macular reflex was exaggerated and mottled. It was Dr. Cutler's opinion that there had been sudden occlusion of the inferior branch of the central artery, which had possibly cleared up somewhat to the nasal side, but which was complete to the temporal side and involved the macula.

The case was referred to me through the courtesy of Dr. Cutler, and first came under my observation on October 9, 1922.

The patient gave the history of having had, in her sixth year, acute rheumatism, and dry pleurisy six years before the present ocular disturbance. An examination of the heart was negative. A specimen of blood submitted for the Wassermann test was also negative. The tonsils were found badly infected and were enucleated. An X-ray examination revealed three apical abscesses, and the affected teeth were extracted. The accessory nasal sinuses were clear. The sella turcica was normal.

Perimetry on October 9, 1922, revealed an almost complete defect of the upper field, as shown in Figure 1. A similar

examination on October 16, 1922, showed return of vision in the upper temporal field, as shown in Figure 2. On November 13, 1922, the fields were again examined and a distinct improvement noted in the temporal projection, although the macular region remained affected. (See Figure 3.)

Details of the perimacular involvement are shown in Figure 4 (Tangent screen at 1 meter; 5 mm. test object.).

Examination of the fundus on November 13, 1922, showed attenuation of the inferior temporal branch of the retinal artery, with a corresponding decrease in the lumen of the inferior temporal vein. The inferior nasal vessels were of normal size. There was distinct relative hyperemia of the macular region. The inferior temporal sector of the optic disc was pale.

When examined on January 24, 1923, there was evidence of great improvement in the macular function, as fixation had defi-

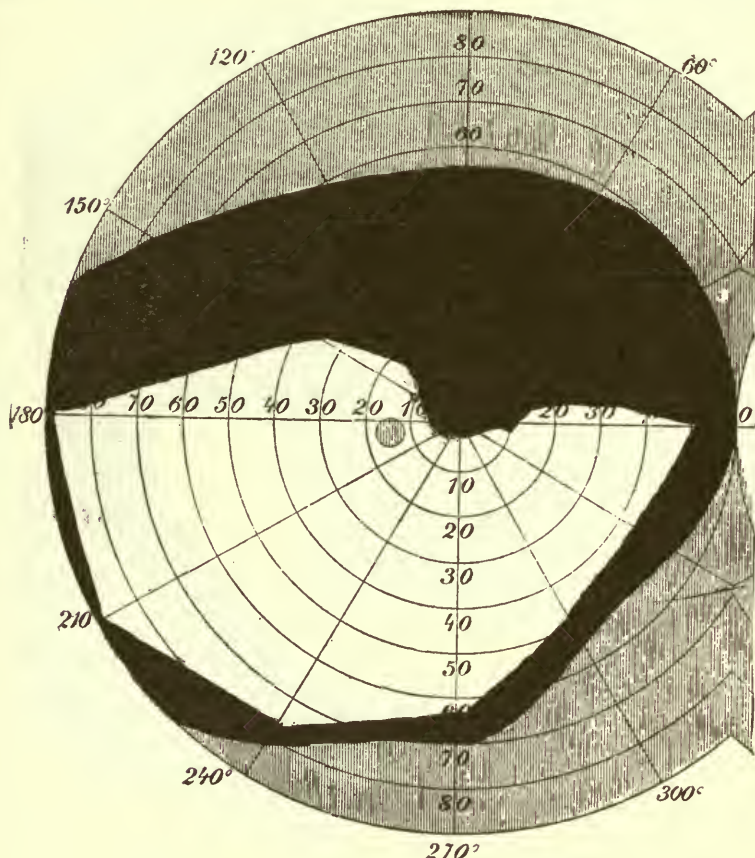


Fig. 1. Field on October 9, 1922.

nately returned. (See Figure 5.) Vision O. S. 6/6—; test type $+.75$ D. read with ease.

In April, the vision had returned to 6/6 (test type $+.50$ D. read fluently), and refraction under a cycloplegic revealed one diopter of latent hyperopia in each eye. At this time, there was definite evidence of periarterial connective tissue proliferation along the course of the inferior temporal artery. Examination of the fields showed no further changes.

The sudden appearance of the field defect, without prodromal symptoms and while the patient was fully conscious, favors the diagnosis of embolism rather than that of thrombosis, particularly in view of the fact that the patient gave the history of an attack of acute articular rheumatism, which may have resulted in a cardiac lesion too minute to be revealed by physical examina-

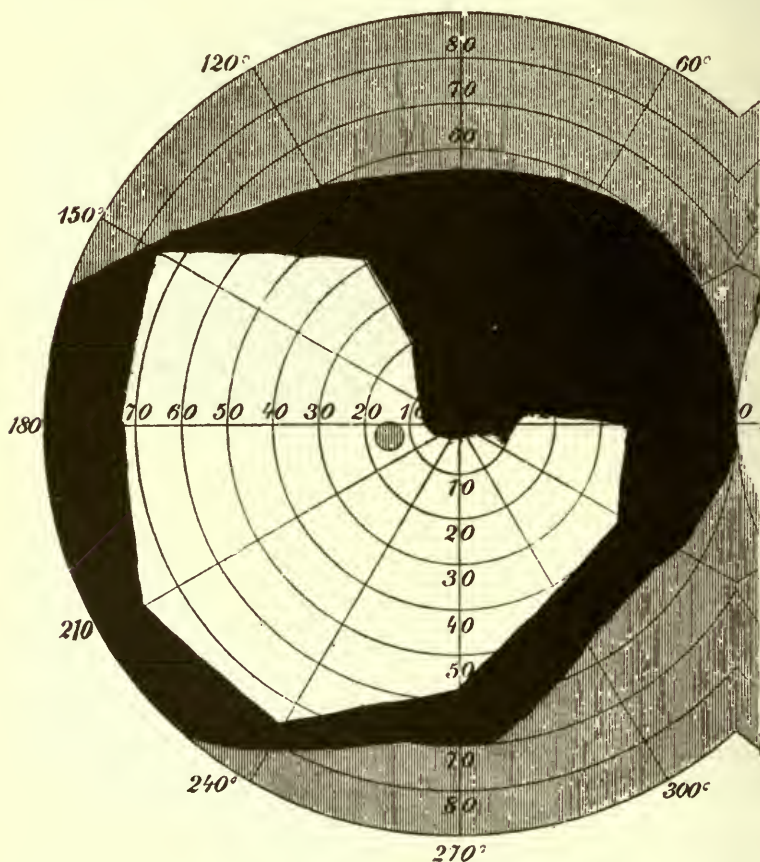


Fig. 2. Field on October 16, 1922.

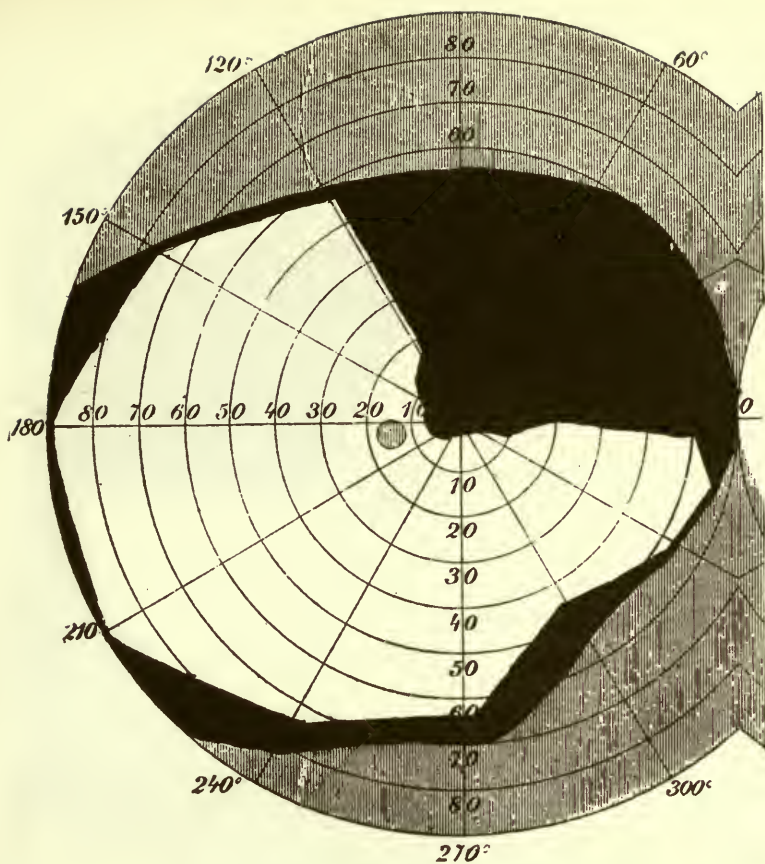


Fig. 3. Field on November 13, 1922.



Fig. 4. Tangent screen. Perimacular involvement on November 13, 1922.

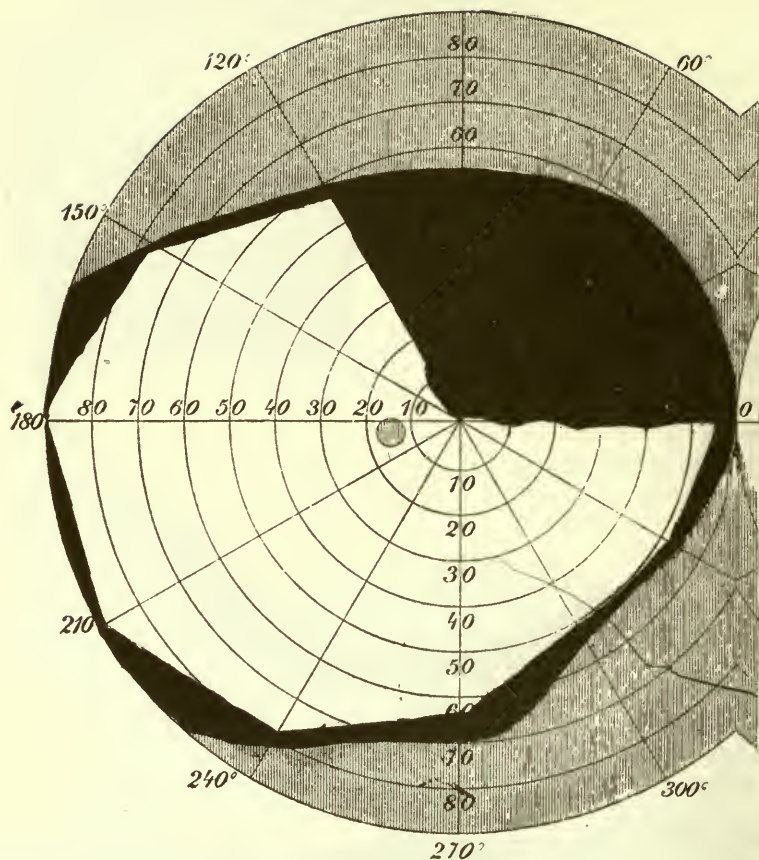


Fig. 5. Field on January 24, 1923.

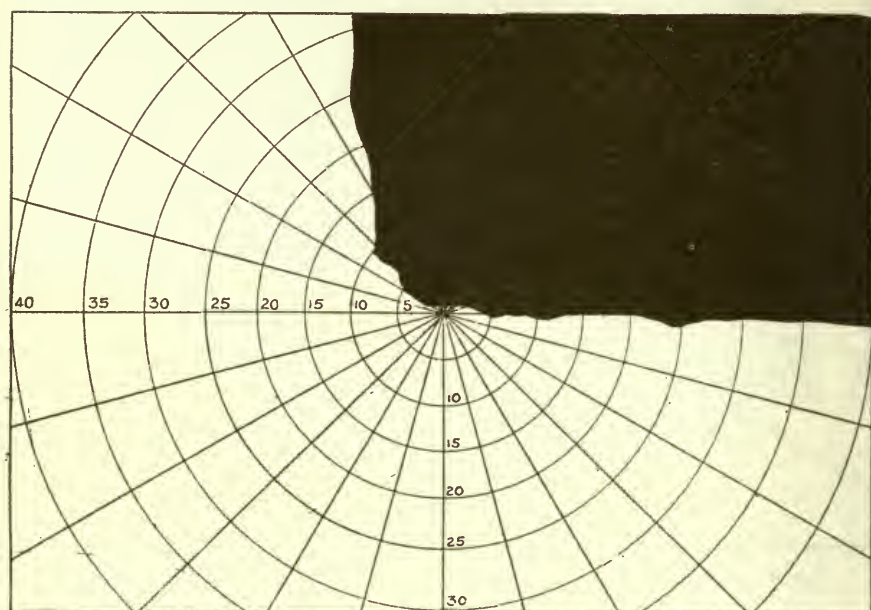


Fig. 6. Tangent screen. January 24, 1923, showing return to central vision.

tion. On the other hand, it must be admitted that thrombosis cannot be absolutely excluded.

Whichever diagnosis is correct, the aspects of the case are strikingly unusual, in that a great part of the upper temporal field remained defective for a period of over ten weeks, and that macular vision did not reappear until fifteen weeks or more after the onset of the ocular disturbance.

The possibility of hysteria might be considered in this case, but this is very unlikely, in view of the absence of characteristic symptoms and in the constancy of the findings obtained with the tangent screen.

In a case of embolism of a branch of the retinal artery reported by Ball¹, there was almost complete loss of the upper nasal and temporal fields, with vision 15/100, four hours after the onset of the disturbance. Twenty-four days after the attack, the vision had improved to 15/50, but without any distinct change in the field. The subsequent history of the case is not given, and it is presumed that there was no complete recovery of central vision as in the present case.

Another case reported by De Schweinitz², with occlusion of the superior temporal artery of the retina by thrombosis, the nasal field remained unchanged, the patient having remained under observation for over two years.

In Veasey's case³ of embolism of the superior branch of the central retinal artery, the lower half of the visual field remained permanently abolished. This case bears some resemblance to the one here reported, in that four days after the attack the vision was 5/22, and that there was return to normal central vision in two months' time. In this case, however, the fixation point remained in the preserved portion of the field.

The general conception of the effect of occlusion of the retinal artery is well expressed by Würdemann⁴. Speaking of embolism and thrombosis he states: "Neither of these conditions is amenable to treatment after they have existed for some days . . ." This statement, of course, is intended to apply only in cases where occlusion is absolute. In the present case, however, it is probable that the occluding agent, whether embolus or thrombus, was of such shape as to completely block the inferior temporal branch of the retinal artery, and to partly interfere with the blood flow in the inferior nasal branch. Through changes in shape or position of the embolus or thrombus, the normal circulation of the inferior nasal branch was gradually restored, and it must be assumed that the decreased flow in the

partially occluded branch was sufficient, from the beginning of the ocular disturbance, to maintain the viability of the retina, although not in itself sufficient to maintain normal function.

On the other hand, the late reappearance of macular function cannot be attributed to any such phenomenon, as the ophthalmic findings indicated the permanent blockage of the inferior temporal branch. Two small ciliary vessels, extending temporally less than a disc diameter from the edge of the optic disc, could hardly have contributed to the late return of macular vision. The most likely explanation is, that the occlusion of the inferior temporal branch sufficiently interfered with the macular function to hold it in abeyance, until sufficient time had elapsed to permit restoration of function by compensatory improvement in the circulation of the superior temporal retinal branch. In this connection, it is important to note that the retinal edema of the inferior temporal quadrant of the retina had disappeared completely when the case first came under my observation, so that edema could not have been a factor in the delayed return of function.

REFERENCES.

1. Ball, J. M. N. Y. Medical Journal, 1895, v. 62, p. 689.
2. De Schweinitz, G. E. Philadelphia Medical Journal, 1903, v. 11, p. 464.
3. Veasey, C. A. American Journal of Ophthalmology, 1919, v. 2, p. 817.
4. Würdemann, H. V. American Journal of Ophthalmology, 1920, v. 3 p. 513.

DISCUSSION.

DR. W. H. WILMER, Washington, D. C.: Many cases of obstruction of the retinal arteries have been reported; but the causes and the pathologic changes are not always clearly recognizable, and the prognosis is usually unfavorable. Therefore, we are indebted to Dr. Smith for his interesting contribution.

Many authors, notably Coats, feel that most of these cases are in reality of a thrombotic nature. The confusion of these two processes is quite natural, considering their essential similarity. In one case, the occluding substance is carried by the arterial blood stream from some distant point, while in the other, it is the result of a local and more gradual obliterating process. Similar conditions predispose towards each type, the clinical symptoms are very much alike, and it is difficult by ophthalmoscopic picture or laboratory examination to differentiate between the two conditions.

However, I feel that in cases of extreme youth, with endocardial or similar lesions, the nature of the eye involvement is truly embolic. For instance, Gruening reported the case of an eight year old boy, who had an organic heart lesion, and Stephenson the case of a girl of eleven years, with a similar cardiac involvement. Moore feels that the embolic process, in general, is not a rare condition. He said that "at times minute emboli occur in the blood stream of individuals in health, or who are at most

but slightly indisposed. Minute emboli may lodge in the vessels of any of the tissues of the body, except the eye—not excluding most parts of the brain and heart—without giving rise to discoverable lesions, or even disturbing symptoms.”

One interesting phase of these cases is the impossibility, in many instances, of assigning any definite cause. Knapp has put this percentage as high as 30, which corresponds closely to my own experience. However, it is instructive to consider some of the predisposing causes. In a truly embolic process, it is necessary to have some focus from which the occluding matter may separate itself. This condition is presented by pathologic conditions of the myocardium and the large arterial trunks, especially the aorta, miliary tuberculosis, altered composition and coagulability of the blood itself, distant blood clots, septic conditions, etc.

Both in the eye and in the brain, embolism is more apt to occur on the left side, owing to the fact that the left common carotid springs directly from the transverse portion of the aorta, while the right common carotid is a branch of the innominate artery.

When the arterial trunk of the central artery bifurcates, just where the vessels make an acute bend—almost at right angles—to follow their new course, especial danger of occlusion from embolism or thrombosis is presented by the two physical factors—increased friction from an angular course, and the sudden diminution in caliber of the arterial stream.

Vessel sclerosis in all cases predisposes to arterial stoppage. A generally low blood pressure, or especially the sudden reduction in case of high blood pressure, lowers the driving vis a tergo of the blood stream. This possible effect is evident when one considers the relation of the ocular to the general arterial pressure. According to Magitot, the normal local arterial pressure is to the normal general arterial pressure as .5 is to 1.0.

I feel that the blood pressure element is a factor in the last case of arterial obstruction that I chanced to see. The patient was a healthy, athletic man of thirty-four. He was slender but well built, and gave a negative family and general personal history. However, during the previous ten years, he had experienced about twelve attacks of temporary blindness involving different portions of the visual fields. These attacks were followed by headaches. The last attack of this type occurred about six months before the present trouble. The present attack was ushered in by sudden and complete blindness in the left eye. After a few hours, the vision began to improve until it became 5/200, eccentric. This condition has remained in statu quo. In this case, the arterial block occurred in the main trunk of the central artery. All physical and chemical tests failed to throw light upon the cause, save the blood pressure, which was 118/60. It is certain that in the case under consideration, the blood pressure was below the standard for his energetic life; for Valvarez finds among the Freshmen of the University of California 45% with a blood pressure of over 130 mm., and 23% exceeding 140 mm.

The retinal arteries have no intraocular anastomosis with any other arterial system, and therefore lack this aid in reducing the gravity of arterial stoppage. But Dr. Smith's patient was most fortunate in having vision restored by the establishment of collateral circulation. Cases

have been reported of arterial plugging, where the arterial blood stream was seen to move in one branch in a normal centrifugal direction and in another branch centripetally; at the same time the existence of collateral circulation was visible.

In addition to this possibility, the patient may have the fortuitous existence of a cilioretinal artery to aid in recovery. According to Elschmig, this anomaly occurs in about 7% to 10% of the cases who present themselves for examination.

In regard to treatment, the logical procedure is to attempt to aid nature either in dislodging the clot, or in increasing the blood supply. We are all familiar with the many suggested remedies. Personally, I have found most help from the iodides internally, nitrit of amyl by inhalation, and locally hot applications, and very firm massage.

In addition to our obligations to Dr. Smith for the presentation of this subject, I would like to express my felicitations upon the happy outcome of this case.

DR. JAMES M. PATTON, Omaha, Neb.: In this connection, I wish briefly to report the case of a man of thirty-eight, who was referred to us July 26, 1923. He stated that the day previously he was working in the harvest field, when he suddenly noticed that he could not see with his left eye. He consulted his local oculist, who dilated his pupil and finding an unusual condition, brought him to us the next day.

His vision on presentation was, right eye 20/10—; left eye very faint light perception from below. The left pupil was evenly dilated with atropin.

Ophthalmoscopic examinations showed a large area of gray edema including the lower outer quadrant of the fundus. The lower temporal artery was nearly empty, but at about what looked like 1 mm. intervals, there were small beads of blood which slowly moved along the lumen of the artery. Similar beads in the branches of the main artery were stationary or nearly so. Other retinal vessels were not involved. Diagnosis: embolism of lower temporal artery, with incomplete closure of the lumen.

The patient was given an inhalation of nitrat of amyl, with vigorous massage of the globe, but with no change in the fundus picture. A marginal subconjunctival paracentesis was then made, allowing the escape of considerable aqueous. Massage and amyl nitrat continued. Two hours later, there was a perceptible increase in the flow of blood in the artery.

The paracentesis was repeated on three successive days, each day showing a decrease in the area of gray edema and increase in the volume of blood in the artery.

A notation, eleven days after the first paracentesis, stated that the vessels were normal in appearance and the edema practically all gone. Vision in the left eye with slight correction was 20/20.

I mention this case to emphasize the value of paracentesis in the treatment of this condition, after massage and amyl nitrat were apparently without effect.

DR. J. G. DORSEY, Wichita, Kansas: I am wondering, since Dr. Patton's talk, whether his case was not starting upward at the time when he saw the flow of these minute particles in the vessels. On the fourth

of March, I saw a farmer, 60 years of age, who said that three days previously, while napping and reading alternately, he suddenly lost the vision of his left eye. The picture of the fundus was that of embolism of the central artery. There was no red spot at that time. At the time I saw him, he could count fingers in the upper temporal region. Nine days afterwards, he returned with the vision the same, but the red spot was distinct. I put some cocain into the eye in order to examine it more carefully, and after fifteen or twenty minutes waiting, I saw the movement that Dr. Patton mentioned, as though the veins were emptying themselves, both the upper and lower temporal veins. As a consequence, the vision at the next visit, a week later, was 4/200, and when I saw him the last time, April 30th, his vision was 20/200 and the vessels had refilled. I have not seen him since, and I do not know the final condition, but the movement of the blood in the vessels evidently had taken place in the beginning.

DR. EDMOND E. BLAAUW, Buffalo, N. Y.: I am very much indebted to Dr. Smith for bringing this paper before us, because I have given some study to the matter of obstruction of blood vessels. His case history shows that neither a thrombosis nor an embolism could have taken place. And there is a third possibility, that it is a spasm of the smaller vessels, which interfered with the circulation. I have seen a case of what I thought was embolism of a vessel, but when I took the instrument of Baillard, it was astonishing how quickly we had arterial dilatation; that case was restored in a week. I think Kraupa (Teplitz) has rightly said that this can take place only in the smaller vessels. He gave papaverin to reduce the condition, and got very good results. These conditions are very well known. E. von Jaeger published an observation fifty years ago (1876) under the title, "Blutstorkung."

We should go over the whole patient to see if there is a high blood pressure, and examine for the possibility of hereditary lues.

THE INTRANEURAL COURSE OF THE OPTIC NERVE FIBERS.

HARRY S. GRADLE, M.D.

CHICAGO, ILL.

There are three main classes of fibers within the optic nerve between the eyeball and the chiasm, that are of great clinical import. First and most important, are the fibers from the macula and the immediately surrounding retina, the area of greatest visual acuity, and hence the most vital in the function of vision. These fibers, from the papilla backward, are grouped into a sharply defined bundle, in marked contradistinction to the remaining fibers. Second, are the fibers from the retina immediately around the optic nervehead, the so-called circumpapillary fibers. These do not form a definite bundle, and hence cannot be classified as sharply as the papillomacular fibers. It is rather a matter of personal opinion on the part of the individual observer, as to where the circumpapillary or peripapillary fibers end and peripheral retinal fibers begin. The demarcation of the projected blind spot depends upon the impulses carried by the circumpapillary fibers. The third class is formed by the fibers from the peripheral retina, which includes all of the retina not mentioned in the two previous classes. This is sharply demarcated from the first class, but as was mentioned, blends imperceptibly into the second class.

The course of the papillomacular bundle within the optic nerve anterior to the chiasm was described at about the same time by Samelsohn and Nettleship and Edmund Walter. Their observations were based upon cases of central scotomata of long standing, in which there had been a degeneration of the papillomacular bundles without involvement of the other optic nerve fibers. Serial sections, stained for nerve degeneration, enabled them to trace the course of the bundle backward from the eyeball, and to this day but little has been added to the information they published in 1882. However, their observations have been confirmed, with slight changes in minor details, that come well within the range of normal anatomic variations, by Uhthoff, Wilbrand and Saenger, and others.

But the exact location within the optic nerve of the circumpapillary fibers is still an open question, as is the course of the fibers from the peripheral retina. The solution of the course of

the latter depends upon the former. There are two main theories concerning the course of the circumpapillary fibers, and both are based upon other than anatomic findings. There has never yet come to autopsy a case with permanent enlargement of the blind spot with circumpapillary atrophy, where the course of the fibers from the retina immediately around the disc could be traced backward. Hence, both theories are based upon visual field and other findings. The first is known as the Leber-Bunge-Fuchs theory, from the three investigators who elaborated it to its present status. According to this, the fibers coming from the retina immediately around the optic nervehead lie in the periphery of the optic nerve between the eyeball and chiasm, roughly en-

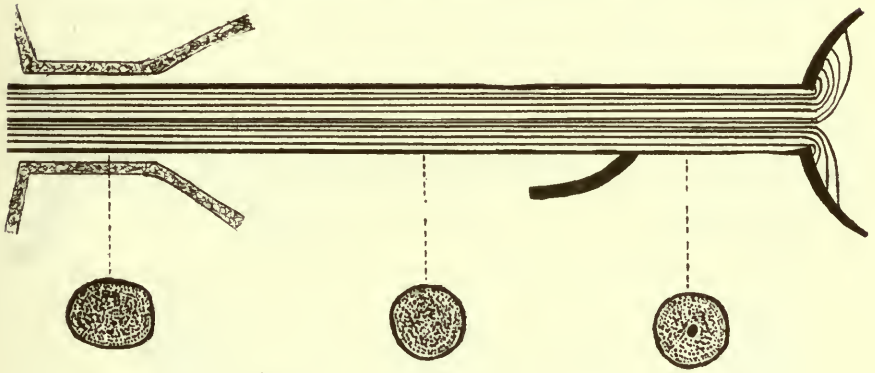


Fig. 1. Leber-Bunge-Fuchs Theory.

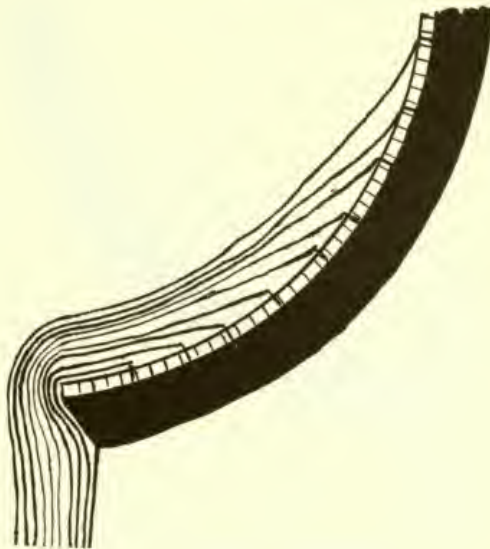


Fig. 2. Leber-Bunge-Fuchs Theory.

veloping the other fibers much as the vaginal sheaths envelop the entire optic nerve. This is illustrated by the diagrammatic sketches 1 and 2. The Uhthoff-Wilbrand-Saenger theory holds that the circumpapillary fibers lie in the center of the optic nerve between the eyeball and the chiasm. It is claimed that these fibers, after leaving the nervous elements of the retina, traverse the entire anteroposterior thickness of the retina. On approaching the anterior limiting membrane, they turn sharply toward the optic nervehead in the course of the other fibers and enter the nerve well toward the geometric center of the disc, remaining in the center of the nerve throughout the entire orbital course of the optic nerve. This is illustrated in the diagrammatic sketches 3 and 4. According to the first theory, the fibers from the periph-

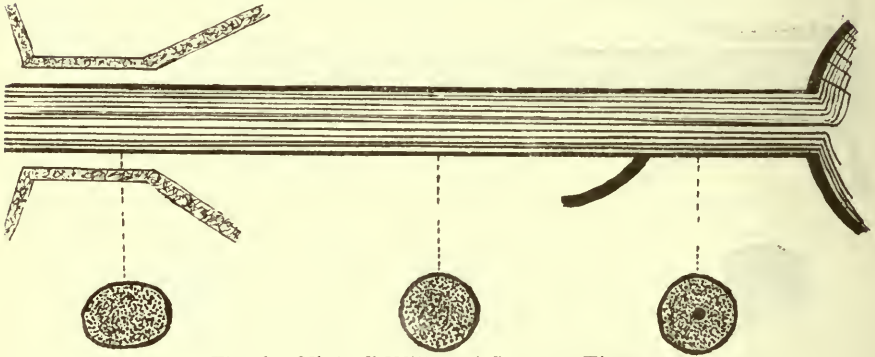


Fig. 3. Uhthoff-Wilbrand-Saenger Theory.

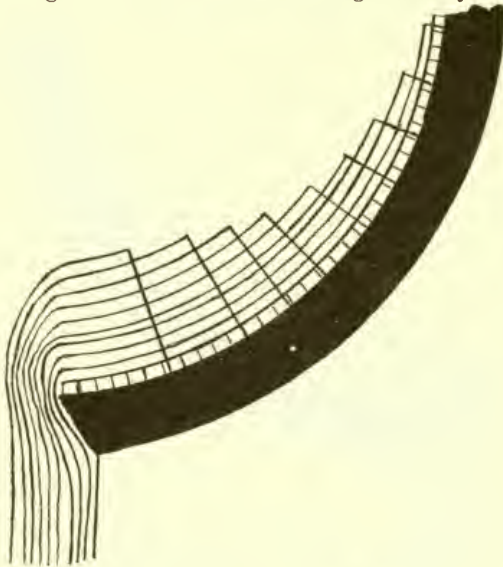


Fig. 4. Uhthoff-Wilbrand-Saenger Theory.

eral retina lie in the center of the nerve and fill the interstices not occupied by the papillomacular bundle and the peripapillar fibers; while the second theory holds that the fibers from the peripheral retina lie in the periphery of the optic nerve and the intermediary section not filled by the papillomacular bundle (in other words, corresponding accurately to the projection of the visual field).

Both theories have had and still have, violent advocates and opponents. Of recent years, the Uhthoff-Wilbrand-Saenger theory has been upheld most vigorously by Igersheimer and Seidel. The former claimed that any scotoma, appearing anywhere within the visual field, was connected to the blind spot by a thin bridge of scotoma. He endeavored to show this by perimetry, radial to the course of the retinal fibers, and accounted for the linear defect by the anatomic findings of Dogiel, who stated that there was a marked decrease in the size of the nerve fiber bundles from the optic disc to the periphery of the retina. Igersheimer believed that this applied to the individual fibers (he forgot that Dogiel said 'bundles'), and that each fiber in its course from the disc to the retinal periphery gave off a minute tendril to every retinal cell that it crossed. Thus the death of one fiber would cause a loss of impulse transmission from every cell to which it was connected, resulting in the scotoma bridge between the blind spot and the peripheral scotoma. But in his last two articles on this subject, Igersheimer has tacitly retracted the majority of this statement. Seidel claims to have proven absolutely the truth of the Uhthoff-Wilbrand-Saenger theory by one bit of experimental work. A patient in the Heidelberg Clinic had an extremely small, peripherally located choroidal sarcoma, on account of which the eye was to be enucleated. The visual field was normal except for the small scotoma caused by the sarcoma. Seidel injected novocain around the optic nerve as near the entrance of the optic foramen as possible, and at intervals plotted the visual field accurately. He found that the peripheral field was somewhat contracted, irregularly it is true, and upon this finding claimed the absolute proof of the theory that the fibers from the peripheral retina were to be found in the periphery of the optic nerve, where their impulses were blocked by the novocain injection. The value of this experiment is negated by the anatomy of the nerve in this area, for here the vascular and lymphatic drainage is toward the center of the nerve, and it is impossible to control the course and extent of the anesthetic.

Of recent years, the Leber-Bunge-Fuchs theory has had two outspoken advocates, Van der Hoeve and Elliot. In an article in which the former showed the fallacies of the Igersheimer theory, he left a very definite impression of his firm conviction in the peripheral location of the circumpapillary fibers. In discussing an early sign in glaucoma, Elliot mentioned the two theories and showed some of the obvious impossibilities of the Uthhoff-Wilbrand-Saenger theory.

In view of the lack of anatomic findings, any point that has a definite bearing upon one or the other theory is of value. The following case, which is the basis of this article, adds one more fact to the foundation upon which the sound Leber-Bunge-Fuchs theory is built.

This anomaly was discovered during the course of a refraction in a man 40 years old. The eye presented no pathologic conditions beyond a slight error of refraction, nor was there any other congenital anomaly to be found. In the left eye, was a band of medullated fibers of moderate density, about $2/3$ of a disc diameter wide. The band became visible in the upper outer quadrant of the disc, apparently arising from the depths of the nervehead in a crescentic line that corresponded with the junction of the middle and outer third of the disc in the quadrant mentioned. The margin of the disc underneath the band could just be discerned as a faint hazy line. The band followed the course of the retinal fibers as usually depicted, out and above the macula, gradually becoming thinner and wider. As the beginning periphery was approached, the medullation became lost, so that in the extreme periphery, no medullated fibers could be discovered. By means of the Gullstrand binocular ophthalmoscope, and by the parallax in relation to the superficial retinal vessels, the depth of the medullated fibers within the retina could be determined fairly accurately. In the peripheric portion, the fibers were fairly superficial, in places covering the vessels entirely, and in others producing an appearance as though the vessels were covered by a delicate veil. This level was practically maintained up to the point where the fibers bent to disappear into the depths of the optic nerve. It was very clear that these fibers, coming from the periphery of the retina, did not lie in the periphery of the optic nerve, at least at its appearance within the eyeball, but that there was a definite area of the optic nerve between these fibers and the edge of the disc.

This finding speaks strongly for the Leber-Bunge-Fuchs theory. If the peripheric retinal fibers were to lie in the periph-

ery of the optic nerve, these fibers should have been extremely superficial in their peripheral origin, but as the disc was approached, should have lain deeper and deeper in the retina and eventually have bent sharply at the edge of the disc and disappeared in the periphery of the optic nerve proper. But this was not the case.

REFERENCES.

- Samelsohn. Graefe's Archiv, 1882, v. XVIII, 1.
 Nettleship and Edmund Walter. Transactions of the Ophthal. Society, 1882-3, v. I.
 Unthoff. Graefe's Archiv, v. XXXII, 3, p. 95.
 Unthoff. Graefe's Archiv, v. XXXIII, 1, p. 257.
 Unthoff. Graefe-Saemisch, II Edition.
 Wilbrand-Saenger. Neurologie des Auges, III Edition.
 Leber. Graefe's Archiv, 1868, v. XIV, p. 164.
 Leber. Graefe's Archiv, 1869, v. XV.
 Leber. Graefe-Saemisch. I Edition.
 Bunge. Habilitationsschrift, 1884.
 Fuchs. Graefe's Archiv, 1885, v. XXXI, 1, p. 177.
 Igersheimer. Heidelberger ophthalmologische Gesellschaft, 1916, p. 343.
 Igersheimer. Graefe's Archiv, v. XVII, p. 1.
 Igersheimer. Heidelberger ophthalmologische Gesellschaft, 1918.
 Seidel. Graefe's Archiv, 1919, v. C, p. 168.
 Dogiel. Archiv f. mikroskopische Anatomie, v. XL.
 Van der Hoeve. Graefe's Archiv, v. XVIIIC, p. 243.
 Elliot. British Journal of Ophthalmology, 1923.

DISCUSSION.

DR. CONRAD BERENS, New York City: The subject of Doctor Gradle's careful study interests me from many angles, but I will limit my discussion to the anatomic side of the question. I have always believed in the theory adhered to by Fuchs, that the peripapillary neurons gave rise to the nerve fibers in the periphery of the optic nerve. This theory appeals to me because I have never been able to see evidence of decussation of the nerve fibers in the retina or optic nerve entrance. It seems to me that the correctness of this theory must be proven on embryologic and anatomic grounds.

I had hoped to be able to find in the collection at the New York Eye and Ear Infirmary some section of the human eyeball with medullated nerve fibers stained by Weigert's method. I regret that I have been unable to find human specimens, but through the courtesy of E. B. Burchell I have found rabbit's eyes and the eye of an elephant. The first specimens of rabbit's eyes showed no apparent crossing of the medullated nerve fibers near the papilla, or in their course through the retina. The same was true of the elephant's eye. I was about to conclude that the case was proven, at least for these two animals, when I found a specimen of a rabbit's eye in which there was an apparent crossing of the fibers at their entrance into the optic nerve. The course of the nerve fibers in all the embryologic sections studied showed such a regular arrangement of the fibers that it seemed hardly possible that there could be any marked decussation.

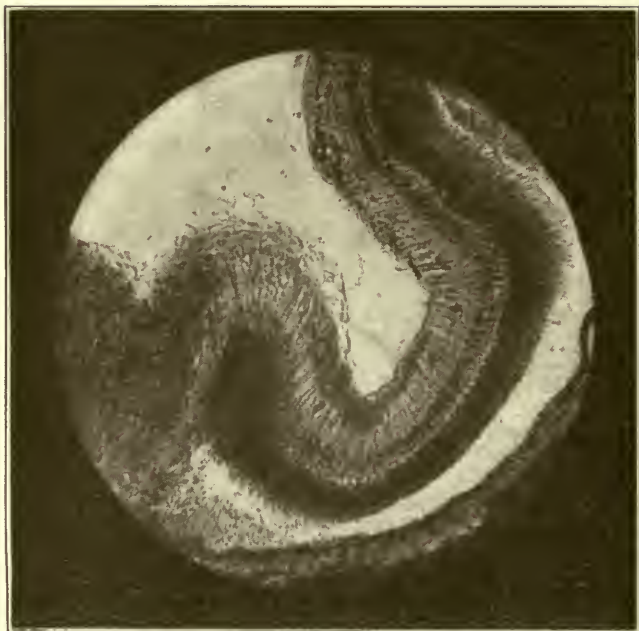


Fig. 1. Medullated nerve fibers of rabbit. No crossing of nerve fibers.



Fig. 2. Medullated nerve fibers of rabbit. A—Fiber decussation.

In spite of the one section found which showed apparent crossing of the nerve fibers at the margin of the disc, which may have been an anomaly, I believe that the embryologic and anatomic evidence sustains the theory of Fuchs upheld by Gradle in this painstaking study.



Fig. 3. Medullated nerve fibers of elephant. No decussation.

DR. LUTHER C. PETER, Philadelphia, Pa.: In connection with Dr. Gradle's case of persistent medullary nerve fibers, it might be well to consider the one published in the second edition of "The Principles and Practice of Perimetry," and previously published by Mr. Basil T. Lang in the Transactions of the Ophthalmological Society of the United Kingdom.

The case which Dr. Gradle has shown tends to confirm our conclusion that the Fuchs theory is correct. In a similar way, the case of Mr. Lang adds materially in the way of confirmation. The medullated fibers form almost a complete circle and stop short of the disc. The two photographs shown together furnish rather conclusive evidence of the Fuchs theory.

From a clinical standpoint, we have two conditions which tend to confirm the correctness of the theory. The first is that of the enlargement of the blind spot, as Van der Hoeve has pointed out, in accessory sinus disease; and the second is that of the differentiation between papillitis and papilledema. Here again the clinical evidence is strong proof of this theory. In papilledema the first field change, and the only change which one will observe if the examination is made early, is enlargement of the blind spots of Mariotte. In papillitis, in which the entire nerve may become inflamed, the evidence is much more pronounced in the peripheral part of the field, exactly where one would expect to find it, along the distribution of the nerve fibers. In papilledema, neither central

macular fibers nor peripheral fibers suffer. You ask why should not the papillomacular bundle be involved at the same time, because the papillomacular bundle occupies the temporal side of the optic nerve? For the same reason that one does not find appreciable central visual loss in glaucoma even though the pericentral area may be much disturbed by a Bjerrum scotoma. So many nerve fibers pass through the papillomacular bundle to the macular area, that one can have destruction of a considerable number of fibers and still have well marked central visual acuity. However, if the papillomacular area were investigated for a variation in light sense, I believe that in all cases of papilledema in which there is enlargement of the blind spot without peripheral change, there would be found some change in the light sense in the macular area. Tests for slight reduction in central visual acuity are not sufficiently delicate to note changes unless marked.

DR. EDMOND E. BLAAUW, Buffalo, N. Y.: Dr. Gradle's presentation is as good as I could imagine. It is, of course, not only of great academic interest that we have a just conception of the course of the fibers in the nerve. Perhaps the two opposing views may be bridged over by a rather intermediate position. This problem has lately been worked out at the Amsterdam University, and published in a doctorate thesis. However, rabbits cannot so well be used, as they have a complete decussation of their optic nerve, in contradistinction to the genus homo.

We must awake the interest of the members in procuring material, which is still of the utmost rarity. The last comprehensive survey of the entire chapter is the work of von Hippel, who takes the stand with Igersheimer, that the section of the macular bundle in the disc is too large as it is at present accepted. That is an interesting topic. For experimentation a very short incision at the periphery of the retina should be made.

DR. HARRY S. GRADLE, Chicago, Ill. (closing): There is more than an academic interest in this subject, for the entire question of the relationship of retrobulbar neuritis of sinus origin and its manifestations in the eye depends upon our interpretation of these theories. If we accept the Leber-Bunge-Fuchs theory, then we will agree with Van der Hoeve; on the other hand, if we do not, we are more or less at sea.

We say the papillomacular bundle is sharply outlined. It is to a great extent. But what constitutes the papillomacular bundle? As Dr. Blaauw said, it shows sharply in the red free light, but if a central scotoma of 2° exists, we would find a difference in the degeneration of the nerve from a central scotoma of 5° or 10° . Consequently, we cannot outline sharply the papillomacular bundle until we determine the exact extent of the macular region.

Sir William Lister, in his admirable address yesterday, presented a case that will require a good deal of study, and it will be interesting to see just exactly how his findings will fit into the subject we have been discussing today. I hope he will make it the subject of an essay at some future time.

A large amount of anatomic research, such as Dr. Berens suggested, will be necessary, but with the passing of time and the assistance of all the members interested, we hope to arrive at some solution of the problem.

CONCERNING SOME GROSS STRUCTURAL ANOMALIES OF THE MUSCLES OF THE EYE AND ITS ADNEXA.

WM. CAMPBELL POSEY, M.D.

PHILADELPHIA, PA.

Functional disturbances of the eye muscles are common, and for the most part easy of recognition by the ophthalmologist; those the subject of this paper, as embodied in the title, are rare, though in all probability occurring much more frequently than is usually supposed.

Positive evidence of the existence, position and structure of any one of the muscles moving the eye or the lids can be obtained only by actually viewing the muscle in question, which naturally is alone possible antemortem at operations, or postmortem by dissection.

In many cases of congenital squint, the supposition of the absence of a muscle arises when there is a total absence of motion of the eyeball in the field of action of such muscle, but such observations are purely hypothetical, being without definite basis. Even at the time of operation, it is impossible to reach any satisfactory conclusion regarding the condition present, for, owing to the conicity of the orbit, the surgeon can see, in the case of the muscles attached to the eye at least, but the proximal end of the muscle engaged. All estimation as to the extent, origin, course, etc. of the remainder of the muscle must be conjectural.

Until the present at least, postmortem dissections of the orbit have been comparatively few. The average undergraduate medical student gives but scant attention to that part of the cadaver, and research in that field has been left pretty much to the anatomists, and apparently but few of these have devoted much time to orbital dissection.

Just as ophthalmic surgeons, however, with large experience have not infrequently encountered marked structural anomalies of the muscles in their squint operations, often as a source of considerable embarrassment at the time and greatly interfering later with the results they had desired to obtain, so the few anatomists who have dissected many orbits have found, not infrequently, marked anomalies in the musculature of the eyes and

lids. In his excellent work on the anatomy of the orbit, Whinnall acknowledges this in the following words: "It is probable, to judge from the writer's individual experience in finding quite a number of gross abnormalities of the ocular muscles in his series of dissections, that such are by no means as excessively rare as would appear from the number recorded in the literature; dissecting room conditions do not favor their identification, and in life some may be unrecognizable through compensatory action of the other muscles."

In describing the few cases of his own which the writer desires to record, it may not be amiss to interpolate a synopsis of the literature, and in so doing to consider the subject under two headings, including (1) supernumerary and anomalous muscles of the lids and orbit, and (2) those of the eyeball itself. Concerning the former, one cannot do better than quote from Lucien Howe's classic work on Muscles, as follows:

"SUPERNUMERARY MUSCLES OF THE ORBIT. So little mention is ordinarily made of the supernumerary muscles, that one not accustomed to dissections may be surprised perhaps that such muscles exist. But anyone who has observed the variations in the secondary insertions in different individuals, or who knows how the bands of connective tissue in different parts of the orbit vary in their distribution, can easily understand how these bands may be sufficiently marked in certain cases to be described as special muscles.

For it must be understood that none of these supernumerary muscles approaches in size the other extrinsic muscles, but consist only of bundles of striped muscular fibers interwoven with connective tissue fibers.

1. *Muscle of Horner.* Among these supernumerary muscles, attention should first be called to the muscle of Horner. This is seldom mentioned in descriptions of the contents of the orbit, yet it is often of considerable importance in connection with the operation for tenotomy of the internal rectus. * * * The muscle consists of a few bands, which arise from the crista lacrimalis posterior and pass horizontally forward and somewhat outward, to be inserted into the tissue just anterior and to the inner side of the caruncle. Farther anteriorly, they pierce the network of connective tissue to be inserted into the conjunctiva and the adjacent structures. The function of this muscle is not well understood. Possibly it is the remnant of the band which moves the nictitating membrane in the lower animals, or it may assist in facilitating the flow of tears away from the globe. How-

ever that may be, the direction of the fibers shows without question that it also tends to draw the conjunctiva inwards and backwards as long as the orbital fascia is in its natural position. While the action of Horner's muscle is thus difficult to see in the normal condition, yet when the fascia is disturbed to any great extent, as in lacerations such as follow certain forms of tenotomy, the fibers of this muscle, then having nothing to counteract them, draw the conjunctival tissue in and backward, and we have a sinking of the caruncle, with the subsequent deformity.

2. *The Gracillimus or Transversus.* At quite an early date, careful anatomists recognized at least one or two other supernumerary muscles—these lying near the roof of the orbit. Albinus was probably the first to describe one of them. He found a band passing from the levator with connections inward, especially to the superior oblique, and this muscle he called the gracillimus. * * *

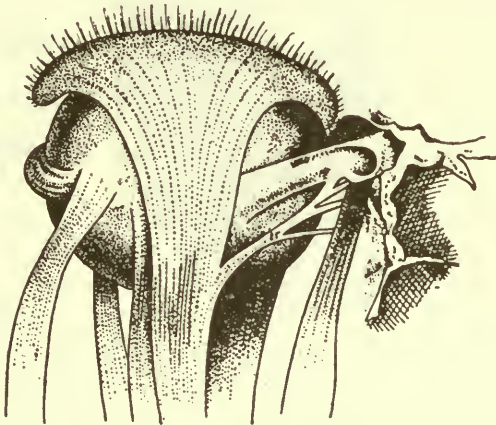


Fig. 1. Supernumerary muscle, the transversus.

A more careful search for the gracillimus, or a muscle similar to it, was made by Budge. He says: "While making preparations of the muscles of the eye, I found a muscular portion given off from the levator palpebrae superioris. This portion branched into two small bundles, from the inner side of the muscle, and then, passing inward, they were inserted into the trochlearis." He examined about thirty orbits of children and grown persons as to this point, and there were only five without some traces of these fibers, although in some instances they appeared to be hardly more than threads.

Among the muscular bands found by Bochdalek in the upper portion of the orbit, he describes another as the "anomalous transversus." (Fig. 1). He says it arises from the anterior

and upper portion of the orbital plate of the ethmoid, and passes almost directly across the upper part of the orbit. At its origin, it consists of small tendinous bands, 3 to 4 mm. in thickness; these enlarging into fleshy bundles, give off various attachments to neighboring fascia, and especially to the levator palpebrae. In fact, when the transversus is small, it forms practically a part of that muscle.

Other supernumerary bands, more or less abundant in muscular fibers, have been met with in the orbit, and are mentioned in the literature from time to time, some of the writers being apparently ignorant of the observations made by others. It is quite certain, therefore, that these additional fibers are more

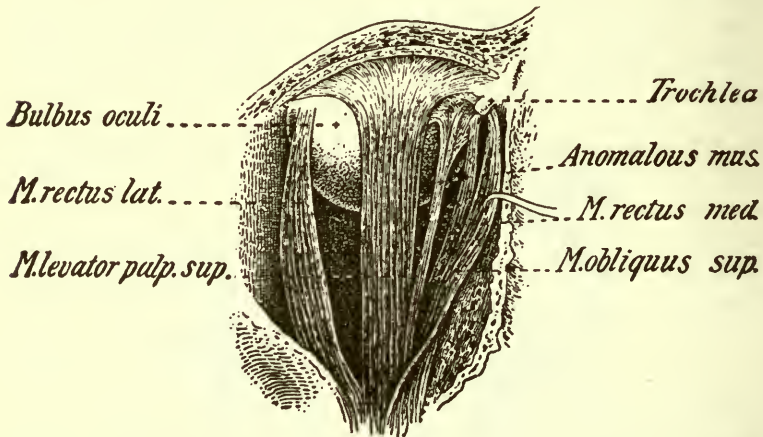


Fig. 2. A drawing of an actual dissection of some of the extrinsic muscles of the eyeball, showing, in addition to the usual muscles, the origin, insertion, and relations of the supernumerary or anomalous muscle.

common than the usual descriptions would lead us to expect. Even in a small collection of orbits, there are usually one or two which illustrate very well the presence of these minute muscular bands. Usually they are what might be termed supporting fibers, which are more or less continuous with the muscle, and which run in the same direction. Sometimes, however, they are quite distinct, passing in the direction, not of the fleshy portion of the muscle, but rather of its tendinous expansions. A good example of this is found in the striated fibers often present in the upper and outer portions of the orbit, in the region where the external bands of the levator bend outward or even partly backward, to be lost in the tissue of the orbit near the lacrimal gland." (Fig. 2.)

In a recent dissection, Rush and Schaeffer found a slender but well defined supernumerary muscle, which they thought was in some respects in agreement with the muscle described by Budge, the insertion of the muscle being, however, more extensive. Their description is as follows: "It is a well defined muscle of from 2 to 3 mm. in thickness, which had a common origin with the levator palpebrae superioris along its medial border. At a distance of 12 mm. from their joint origin, it parted company from the levator, and passing forward, it assumed a position in the orbit almost equidistant from the obliquus superior, the rectus medialis, and the levator palpebrae superioris. At a distance of 30 mm. from its origin, the muscle spread out into a peculiar fan shaped attachment. The stronger central fibers of the fan extended forward in two or three strands to be attached to the upper and lower borders of the trochlea, and even to the frontal bone beyond. A lateral expansion of the fan was directly continuous with a similar expansion from the levator, and an inferior expansion was attached to the superior border of the rectus medialis. It received nerve filaments from the branch of the oculomotor nerve which supplied the levator. Traction upon the muscle gave a feeble internal movement of the eyeball."

In inquiring into the causes of congenital ptosis, Briggs noted the following underlying conditions at fault.

"(a) *Defective development of levator and other muscles of the eye.* Heuck found a partly developed levator measuring only 2 mm. in breadth. Bach, in a case of bilateral congenital ptosis and limitation of the eye movements upward, found defective development of levator and moderate atrophy of superior rectus; the nuclear region of the oculomotor was normal.

(b) *Adhesion of Muscles.* Albers and Wrisberg found, in a case of ptosis, adhesion of the rectus superior with the levator; the external rectus was adherent to the inferior rectus, and the internal rectus to the superior oblique.

(c) *Abnormal insertion of muscles* was found by Rossi, Heuck, Dieffenbach, Pflüger. In some of the cases the superior rectus was found inserted back of the equator.

(d) *Connective tissue bands instead of muscles* were found by Ahlström in cases of congenital ptosis. When he laid bare the tarsal border in the left eye, no trace of the levator tendon was seen, and in the right eye only a few scattered tendon fibers were found.

(e) *Absence of muscle.* Lawford, in a case of ptosis with divergence, found the rectus internus absent. Ahlström found no trace of the levator, and Heuck reports the same condition. Harles cites a case of ptosis in which both obliques were missing, the recti being normal. Seiler found a case of ptosis in which the inferior oblique and superior rectus of the right eye, and the inferior oblique of the left eye were lacking. In another case, the superior and inferior obliques of the right, and the superior and inferior obliques and superior rectus of the left were not found.

Steinheim, in a case of congenital ptosis with defective motion of the eye, failed to find the superior rectus."

Anomalies of the Musculature of the Eyeball Itself. The literature contains a number of accounts of anomalies of muscles governing the movements of the globe itself—some quite complete, others more or less fragmentary. The majority relate to the absence of a muscle. Thus, Krause, Ledouble and Lawford noted absence of the internus; Krause and Bahr of the externus; Seiler and Steinheim of the superior rectus; Stieren and W. E. Davis of both inferior recti; Coover of the superior and inferior recti; Hartes and Seiler of the obliques. Absence of all the eye muscles is described by Klincosch.

In operations for the correction of congenital strabismus, the writer, in not a few instances in his own cases and those of his colleagues, has noted an apparent complete absence of the rectus muscles, in other instances much attenuated muscles, and in still others the replacement of the muscle by a fibrous band. Thus, in one case of defective downward motion in the right eye recently operated on by him, he was unable to find a trace of the inferior rectus muscle. The anomaly occurred in a young man, the left side of whose face was underdeveloped; with the left eye fixing horizontally, the right eye deviated strongly upwards. The right eye could not be moved down and out, although it could be turned by the superior oblique somewhat down and in. At operation, the inferior rectus was found to be absent, except a very rudimentary portion at the usual site for its insertion. To this, the lower halves of the internal and external tendons were sutured, and a free tenotomy done on the superior rectus, care being taken to bring forward as much as possible the lower part of the capsule of Tenon. At the end of two weeks, the eyes were directed on the same plane and there was a left hyperphoria of 10°. (A. J. O. 1921, v. 4, p. 524.)

Muscles merged together or with union of neighboring parts

have been reported by Olbers and Wrisberg. Morgagni recorded a union of the superior oblique with the trochlea, while Macalister found the fascia which often binds the rectus internus and inferior at their origin, to be extended, so that both these muscles were united from the posterior third of the orbit. The external rectus may be inserted into the globe by two tendons, as found by the writer, in the eye of a boy during a squint operation. (Fig. 3.)

The right eye had converged from birth and glasses, though worn since six years of age, had not corrected the deformity. Examination showed an alternating concomitant converging squint of 45°. A double advancement was performed, at which

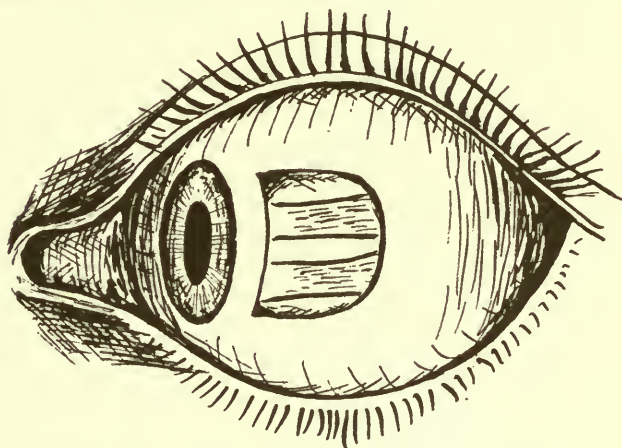


Fig. 3. Bifurcation of external rectus muscle.

time, in place of the single insertion of the left externus, the muscle was found to be inserted into the globe by two tendinous bands, symmetric in size and development, and about 4 mm. apart. The tendons blended into two well developed muscular bellies, which ran parallel to one another until they were lost posteriorly in the tissues of the orbit. Whether they continued as separate muscles to the apex of the orbit, or formed a common belly before arriving at this position, was of course impossible of ascertainment. The right externus was inserted normally. The results of operation by the usual twin mattress sutures were satisfactory.

This case is somewhat analogous to Wicherkiewicz's, who found while performing an advancement of the internal rectus, that the muscle was inserted by two tendons, separated vertically from each other by a distance of 4 mm. Dissection revealed that this space decreased posteriorly so as to form a triangle, with

its apex in the belly of the muscle, 16 mm. from the line of insertion. Macalister, whose treatise on anomalies of the ocular muscles is one of the most interesting on the subject, observed the external rectus supplied with two heads. Dieffenbach and Behr also noted instances of bifurcated muscles, while Zagorsky and Albinus found double external recti muscles.

A lateral rectus with two extra fasciculi, which passed forwards to terminate in the inferior tarsal plate and lateral wall, is recorded by Curnow.

In one of his specimens, Whitnall found a well marked fleshy bundle, 7 mm. long and 2 mm. in diameter, passing from the lateral rectus across the posterior third of the orbit, beneath the optic nerve, to fuse with the belly of the medial rectus; no nerve could be traced to it.

Gross deviations in the location of the insertion of the tendons have been recorded by Rossi, Dieffenbach, Pflüger and others. Ter Aruntinjan, at an operation for strabismus, found the internus tendon very broad and adherent to the sclera by an insertion above the normal one. The superior rectus was very strongly developed, while the inferior rectus consisted only of a feeble tendinous bundle.

In advancing the left external rectus, Bourgeois found no trace of it at its usual insertion, but discovered it inserted far superiorly and in an oblique direction. The muscle was much atrophied, scarcely 2 mm. broad.

In a case of retraction of the eye, Türck noted absence of the rectus externus. The internus was inserted 12 mm. from the corneal limbus, and by a second head still more posteriorly, which accounted for the retraction.

The superior rectus was found by Aubaret to give off a muscular slip 15 mm. long, which arose from the same origin from the annulus of Zinn and passed downwards and forwards across the lateral face of the optic nerve to join the inferior rectus about its midpoint; the nerve supply came from the inferior division of the third nerve.

In a preparation studied by Whitnall, the inferior rectus could be seen giving off a large muscular bundle, which passed lateral to the optic nerve and joined the superior rectus; it was innervated by the lower division of the third nerve.

In his text book quoted above, Whitnall states that "the superior oblique may be closely accompanied by an offshoot from the levator palpebrae superioris, sometimes called the "comes obliqui superioris". In one of his preparations, there were

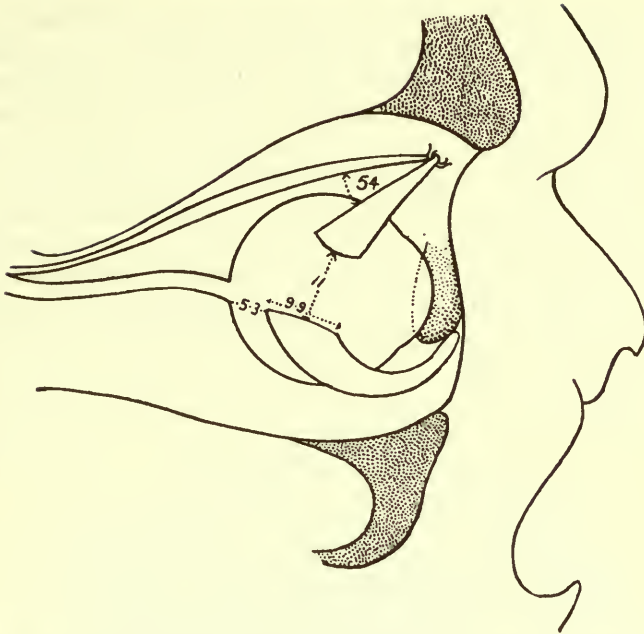


Fig. 4. Diagram to show normal position of origin of the inferior oblique muscle and its insertion onto the eyeball.

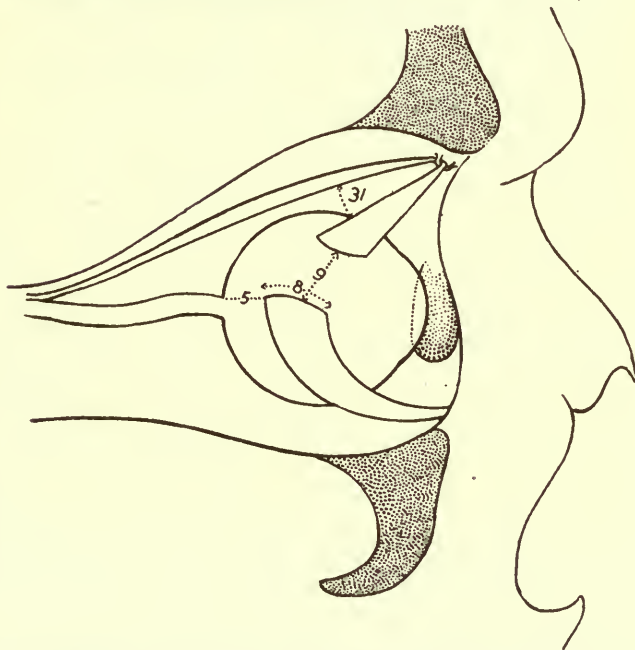


Fig. 5. Diagram to show an instance of abnormal origin of the inferior oblique muscle and its higher attachment onto the eyeball.

present two long muscle fibers, arising in common with the levator and ending anteriorly, the one upon the fascia bulbi between the superior oblique and the globe, the other on the orbital margin beneath the pulley; the nerve supply came from the fourth nerve; the superior oblique was broader than usual. Ledouble has found supernumerary fasciculi accompanying the reflected tendon, and has further recorded a case where the direct or normal fleshy part of this muscle was absent, the reflected or usually tendinous part being muscular and arising from the site of the pulley, recalling the type normally found in nonmammalian vertebrates."

The inferior oblique was found by Whitnall to have an abnormally placed origin in several instances. Out of 100 orbits examined by him, the origin was in the usually described position immediately adjacent to the incisura lacrimalis of the maxilla in

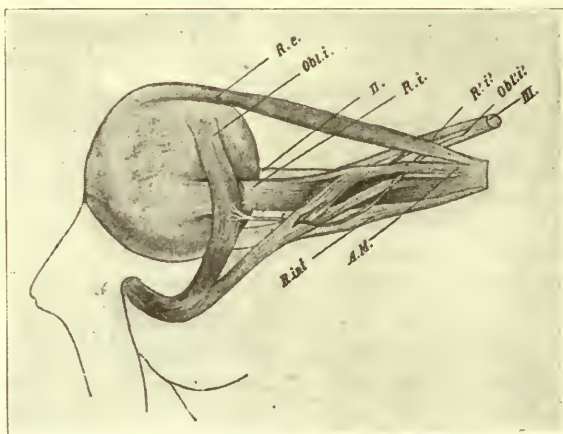


Fig. 6. An accessory inferior oblique muscle.

45 instances; it lay from 2 to 5 mm. distant in 47 cases (most often in the left orbit), and was situated from 6 to 7 mm. lateral to the normal position in 8 cases. Of the latter, the muscles were examined in three eyeballs and found to be inserted higher up than usual, and their total length was normal; the position resembles that found in certain fishes. (Figs. 4 and 5.)

Rex described an abnormal muscle bundle (musculus obliquus accessorius inferior) binocular, which passed from the apex of the orbit to the inferior oblique, sending a slip to join the inferior rectus; both were supplied by the third nerve. (Fig. 6.) This abnormal muscle of slender proportions had an origin from the orbital apex close to that of the rectus inferior and externus,

which blended with the muscle belly of the former and accompanied it to the anterior portion of the orbit. Its insertion with the normally sized and inferiorly situated oblique was through the medium of a broad fleshy band of muscle fibers, about 6 mm. from the point of origin of that muscle.

In correcting a case of right convergent squint of high degree in a young man, which had dated from birth, upon exposure of the tendon of the external rectus muscle, the writer discovered an accessory muscle just inferior to it. This muscle, which was inserted into the globe by a short, rounded tendon, about 4 to 5 mm. long, immediately below that of the externus, was well de-

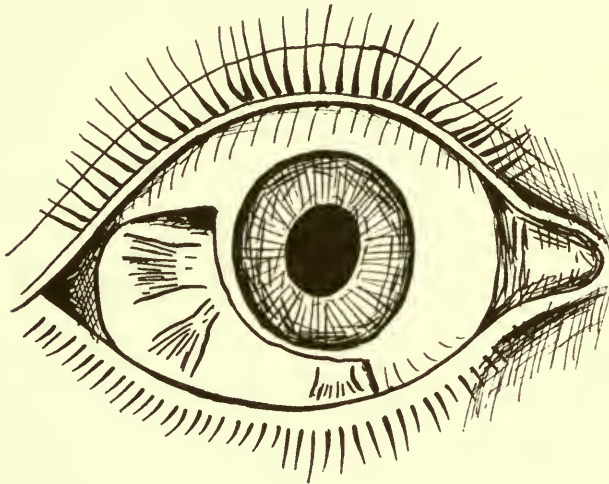


Fig. 7. An anomalous muscle, in association with external rectus.

veloped, more or less rounded, and took an oblique course inferiorly, coursing midway between the externus and inferior rectus muscles, until lost in the tissues at the equator of the globe. Both the external rectus, which was somewhat attenuated, and the anomalous muscle were freed from their insertion and advanced by two twin mattress sutures. The result of the operation was excellent, parallelism of the visual axes being obtained. (Fig. 7.)

In a case of convergent strabismus observed by Baumgarten, the muscle itself was normal but enveloped in a dense connective tissue. Uhthoff saw, in place of a rectus internus, a normally inserted band of fully developed connective tissue. In a case which showed complete left lack of abduction, Bernheimer found a thin band of connective tissue in place of the externus. Axenfeld

reported a similar case in a young man, who was unable to abduct the eyes to the left, although there was binocular vision in looking straight ahead and in the right field of vision. Other instances of replacement of the externus by a more or less inelastic cord have been reported by Baumgarten, Inouye, Alling, Schürenberg and Evans.

Vestiges of the *musculus retractor bulbi* (*retractor oculi*, superior oculi, rectus posterior, choanoid) muscle have on rare occasions been recorded in man, and what would appear to be a well marked example was recorded by Whitnall. (Fig. 8.) To use his words: "The muscle is first met with in amphibians and certain reptiles, and exists in great greater number of mammals, reaching its highest development in the ruminants; vestiges occur

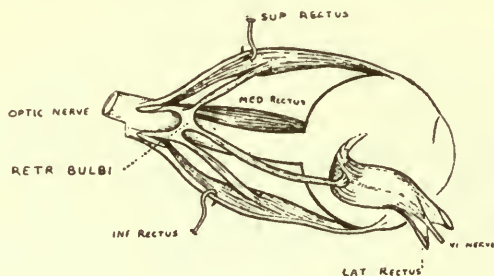


Fig. 8. The retractor bulbi muscle in the right orbit. The lateral rectus muscle has been cut from its two heads of origin and turned forward, thus exposing the tendon of the retractor bulbi, with its four muscular slips passing to the four recti muscles. The third and fourth nerves passed between the sheath of the optic nerve and the tendon of the retractor bulbi, the latter separating these nerves from the sixth.

in some monkeys (e.g. *macacus rhesus*). The muscle arises from the apex of the orbit, lies wholly within the cone of muscles formed by the recti, and is inserted onto the posterior hemisphere of the globe; it may exist either as a cone shaped mass cleft only by a passage for the optic nerve, as in the pig, or be subsequently divided into two or more parts. In the cat, for example, the muscle is divided about 2 mm. from its origin into four slips, which diverge and pass forwards to be inserted onto the globe behind the attachments of the recti and opposite the intervals between them. It is to be regarded as a derivative of the lateral rectus, and is innervated in the domestic animals by the sixth nerve, though in the writer's example in man the third nerve shared. Its action is antagonised by the *membrana orbitalis muscosa* or protractor of the globe. In the macaque monkey, it is found reduced to a

single bundle placed between the superior and lateral recti, in which position Ledouble found delicate muscle bundles in two cases in man. Other instances have been noted by Nussbaum, and Fleischer (1907), and according to Lewitsky (1910) it is always represented in man (thirty specimens examined) by a "well marked strand of connective tissue (fascia retrobulbaris), lying between the lateral rectus and the optic nerve, and attached to the back of the fascia bulbi anteriorly; it does not appear, however, to be readily indentifiable."

According to anatomists, all these anomalies which have been cited are to be explained by intrauterine errors in development, by cleavage from the common premuscular embryonic mesoblastic mass.

CONCLUSIONS.

The foregoing is a fairly complete record of the gross anomalies of the muscles of the eye and its adnexa contained in the literature, and in the opinion of the writer possesses more than an historic interest, and is worthy of more than a hurried survey. All must have been accompanied by deviations in the ocular axes which were aberrant and confusing to an examining clinician, and perplexing and baffling to an operator desirous of straightening the eyes. Many cases of congenital squint are dependant upon such anomalies, and it is only by an appreciation of this fact and a better knowledge of these peculiarities, that the surgeon by modifying his plans can overcome the difficulties in operating they entail and can succeed in establishing parallelism of the visual axes.

More frequent and more careful dissections of the human orbit are necessary. Ophthalmologists should familiarize themselves, also, with the orbital anatomy at least of the lower animals, for many of the anomalies enumerated will be found to be but reversions to lower types and not purely of adventitious occurrence.

BIBLIOGRAPHY.

- Allis, Phelps. *Anat. Anzeiger*, Bd. 16.
 Aubaret. *Rec. d'Opht.*, xxii, p. 220.
 Bahr. *Bericht ophth. Gesch. Heidelberg*, No. 25, p. 334.
 Bourgeois. *Recueil d'Opht.*, 1891, p. 708.
 Bowditch. *Journal of the Boston Soc. of Med.*, 1898, ii, p. 224.
 Briggs. *American Jour. of Ophth.*, 1919, vol. 2, p. 408.
 Budge. *Zeitschrift für rationale Medizin*, 1859, 3 vii, S. 273.
 Coover. *Trans. Colorado Ophth. Soc.*, Feb. 20, 1909.
 Crisp. *Annals of Ophth.*, vol. 33, p. 587.

- Curnow. *Journal of Anat. and Physiology*, June 1873, No. xii.
 Green. *Amer. Jour. of Ophth. and Oto-Laryngology*, 1913, p. 358.
 Heuck. *Klin. Monatsbl. f. Augenheilkunde*, Bd. xviii, 1879.
 Hoover. *Trans. Colorado Ophth. Soc.*, Feb. 20, 1909.
 Howard. *A Practical Handbook on the Muscular Anomalies of the Eye*, 1899.
 Howe. *The Muscles of the Eye*. 1907.
 Krause. *Archiv für Augenheilkunde*, 1912, vol. xxi, p. 977.
 Ledouble. *Arch. d'Ophth.*, T. xiv, p. 218, 1894.
 Macalister. *Trans. Royal Irish Acad.*, 3rd series, vol. 25, p. 7.
 McDonald. *Arch. of Ophth.*, vol. 43, p. 515.
 Millendorf. *Manhattan Eye and Ear Hosp. Rep.*, vol. xv, 1914, p. 1914.
 Poyales. *Anatomical Record*, 1917, vol. 13, No. 6, p. 375.
 Pflüger. *Klin. Monatsblätter für Augenheilkunde*, xiv.
 Rush and Schaeffer.
 Thomas. *Lancet*, p. 1442, 1896.
 Whitnall. *Anat. of the Human Orbit*, p. 283.
 Wicherkiewicz. *Klin. Monatsbl. f. Augenheilkunde*, Feb., 1907.

DISCUSSION.

DR. LUTHER C. PETER, Philadelphia, Pa.: The subject matter so well presented by Dr. Posey is of more than passing interest. As the essayist has pointed out, muscular anomalies are probably of much more frequent occurrence than is usually supposed. It is not unlikely that many cases of abnormal motility may be due to anomalous attachments, anomalous origins, and especially to anomalous fasciculi or bifurcations. Clinically, they are not very frequently observed, although possibly often overlooked. The abnormalities encountered in the anatomic room would lead one to believe that more should be recognized on the operating table. There never has been any good surgical reason advanced for concealed or subconjunctival operations on muscles, whereas the open operation, or the exposing of the tendon to be operated on, has a sound surgical basis. Furthermore, if a tendon is carefully exposed, abnormal attachments and fasciculi are more apt to be discovered if present.

Abnormal attachments are probably more frequently observed than accessory fasciculi. According to the cases reported in the literature and observed in the operating room, total absence of a muscle or rudimentary development is exceedingly rare. This relative infrequency, however, can in part be accounted for by the fact that so few congenital palsies are operated on. The results as a rule are not encouraging by the usual methods of advancements and resections, and many, therefore, never reach the operating table, where alone the diagnosis can be definitely made. Unusual development, bifurcations, and anomalous fasciculi are encountered with more relative frequency.

Dr. Posey has referred to many of these abnormalities as reversions to lower types. The studies of Whitnall and some of the cases reported in the literature tend to confirm this observation. An abnormal fasciculus from the superior to inferior rectus, and from external to internal rectus is usual. Bundles of muscle fibers from the external to the inferior rectus, to the orbital wall, and to the lower lid are described by Whitnall. Last spring, the writer encountered an unusual development of the external rectus. The case presumably was one of paralysis of the external rectus in a woman of forty-five years of age, the condition dating from childhood. Clinically, it had all the appearance of a paralytic

internal rectus. An O'Connor operation was undertaken. Exposure, however, of the tendon of the externus revealed an anomalous condition. The muscle was unusually large. Its attachment spread out in a fan shaped fashion, and extended up to the insertion of the superior rectus. Division of the entire insertion corrected the deformity and restored the eyes to parallelism.

The subject, as presented by the essayist, has a broad clinical application. These anomalies undoubtedly enter into the production of at least some of the ordinary forms of squint, and possibly are the disturbing factors in some of the unusual types, which do not follow the usual behavior of the clinical forms with which we are familiar.

With the so-called retractor bulbi, referred to by the essayist, the writer has had no personal experience. It, however, in all probability is a factor in some cases of the retraction syndrome, in which there is a retraction of the eyeball in attempted adduction. I have not found any reference in the literature to this muscle as a causative factor. According to Lewitsky, the muscle is always represented in man "by a well marked strand of connective tissue." It is within the range of possibilities, therefore, that a more than usual development of this muscle, so commonly observed in the lower animals and even in monkeys, may play a dominating role in the retraction phenomenon referred to.

There are types of abnormal motility which are puzzling. They do not correspond to the cause and effect relations with which we are familiar. Some of these cases may have their basis in accessory muscle bands or fasciculi.

Dr. Posey has wisely suggested more frequent dissections of the human orbit, as well as the orbit of the lower animals, as a means of detecting the frequency and character of muscular anomalies found. In this the writer heartily concurs. In addition, however, it is evident that more careful observations on the operating table will also add materially to our knowledge of causation in these interesting anomalies of muscular motility.

THERMOPHORE THERAPY OF EXPERIMENTAL SARCOMA.*

LAWRENCE POST, M.D.

ST. LOUIS, MO.

By studies on the effects of heat applied to the eyeball, it was found that different tissues were affected in varying degrees. For example, the cornea was found to be relatively resistant, while iris and ciliary body were easily destroyed, it being possible to completely eliminate areas of the latter two tissues without damage to the cornea other than a gray infiltration of the substantia propria. It had also been observed by Shahan¹ that epitheliomata were much more vulnerable than normal tissue. It, therefore, seemed probable that neoplasms of the iris and ciliary body might be destroyed without serious damage to the visual apparatus.

It was to demonstrate this that the experimental work to be described in this report was performed.

The most convenient tumor for this purpose appeared to be a rat sarcoma, the so-called Jensen sarcoma, of well known properties. This is a spindle celled sarcoma which originally metastasized, but the transplants from which have lost this property, though retaining the properties of rapid growth and malignancy. These tumors were tested for bacteria with negative results by Dr. Montrose T. Burrows, who has studied this sarcoma extensively and kindly furnished the material for this work.

From a young tumor, a small superficial piece was aseptically removed and the specimen macerated in normal saline. About one-half minim of this suspension was at once inoculated into the anterior chamber of each of a number of albino rats. In several cases, there was a deep infection following the inoculation. This was found to be almost always avoided if the maceration was very fine and a very fine needle used. In successful cases, the first change was noted in about three weeks, at which time a gray infiltration of tumor cells at one or more points in the iris was visible. Usually, the filtration angle was the point of origin of the growth, though occasionally the initial lesion was elsewhere

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in the iris. If not treated the tumor grew rapidly, so that within about one week it entirely filled the eyeball and produced great enlargement of the same, with rupture of the globe and further extension of the tumor throughout the orbit. In one rat which was permitted to survive, the ulcerated and secondarily infected tumor mass measured three cubic centimeters.

A tumor of $1 \times 1\frac{1}{2}$ millimeters in surface diameter was selected for heating. This sarcoma appeared as a definite cream-gray, solid mass, obliterating the iris angle and extending about two-thirds way to the pupillary border. The red reflex of the fundus was easily seen through the transparent iris of the albino rat. The neoplasm was fairly sharply outlined as an opaque spot against this red reflex.

The thermophore was set at 160° . The rat was etherized and a thermophore conductor, with contact surface just sufficiently large to cover the tumor, was applied, with moderate pressure, exactly over the neoplasm. Firm contact with the cornea was maintained for two minutes, the conductor being kept at 160° . The immediate result was a loss of corneal epithelium and a cloudy swelling of the substantia propria of the cornea.

On the second day there was considerable chemosis and redness of the conjunctiva. The corneal epithelium had been replaced. The gray infiltration began to subside in about a week. Soon thereafter, it was evident that the tumor mass was much smaller than at the time of heating. There was a gradual diminution in size of the neoplasm, until it finally appeared as an ill defined gray area in the iris. When the animal was sacrificed for sections, five months after treatment, there was only a very thin gray infiltration of the cornea and of the iris at the limbus under the point heated. Except for this slight clouding of the cornea, which did not extend more than a millimeter from the limbus, the cornea was entirely clear. The anterior chamber always was well filled with aqueous. After the first few days following the heating, the iris and the interior of the eye could be readily seen. Sections through the area occupied originally by the tumor showed a replacement of normal corneal tissue at the limbus by fibrous tissue. The iris and ciliary body at the point heated were thinned, and there was nowhere evidence of tumor cells.

A larger tumor from the filtration angle, filling about one-half of the anterior chamber, was heated with a contact surface which covered only about one-half of the surface of the neoplasm. This

treatment did not materially retard the growth, which soon filled the globe and orbit.

An attempt was made in a similar large sarcoma to cover the entire tumor with the contact surface, but this was found to be such severe treatment that the eye was irreparably damaged. It seemed, therefore, that if the new growth had progressed further than about two millimeters, it was necessary to use such a large contact surface to destroy the tumor, that the integrity of the eyeball was lost.

In rats, a single application of a contact surface large enough to cover the tumor, and not more than two millimeters in diameter, at 160° for two minutes, while usually sufficient to eliminate the neoplasm never resulted in more than a clouding of the cornea. In one case, a second application a week after the first was found necessary. Such second applications, if not made in less than a week, were found to be well tolerated by the cornea.

It is possible that higher temperatures for shorter periods, or lower temperatures for longer periods, might be more efficacious. One hundred and sixty degrees for two minutes was used because it had been demonstrated in other thermophore experiments by Shahan², that the 'maximum degree of heat is not reached in the anterior chamber until the end of two minutes, and other experiments led me to believe that a rat's cornea will not tolerate much greater heat than 160° for two minutes without permanent damage. Shahan showed that 145° for one minute would destroy surface epithelomata. But 145° even for two minutes has little visible effect on the iris, and was thought probably inadequate for elimination of iris sarcomata.

Further tests will be made to determine the optimum temperature.

In all our work we have found the human eye less easily damaged than the eyes of animals, so that it is likely that proportionately larger tumors in man than in rats could be treated safely. In rabbits we have found that the eye would tolerate as high as 200° of heat, applied directly with a contact surface 3x5 mm for two minutes, without destruction of the globe, and if applied at the limbus apparently without interfering with the vision.

There is such a similarity between the experimental sarcoma and the human sarcoma, that I believe that the latter would react in a like manner to the rat sarcoma, but doubt whether the procedure would be justifiable in any but a patient who refused operation. Such a case has not presented itself since undertak-

ing this work, but if this did happen, I should not hesitate to make the attempt to destroy the neoplasm with the thermophore.

BIBLIOGRAPHY.

1. Shahan, W. E. Thermophore Treatment of Ocular Neoplasms. *The Journal of the Missouri State Medical Association*, Nov., 1921, vol. xviii, p. 392.
2. Shahan, W. E. Effects of Heat on the Eye. *Journal A. M. A.*, Aug. 5, 1916, vol. lxvii, pp. 414-417.

DISCUSSION.

DR. JAS. M. PATTON, Omaha, Nebr.: The destruction of new growths by the application of heat in one way or another has long been a common practice, and ranges from the heated fragments of stone of the Aborigines to the modern electrocautery and electrocoagulation.

All methods are more or less subject to the same objection, viz., that we are dealing with an unmeasured and often only partially controlled quantity of heat. In certain cases this was of little moment, for instance when cauterizing a new growth in some indifferent part of the body, where a difference of an inch or so was of no particular moment. But in dealing with a tissue like the cornea, where every mm. is precious, there was a demand for a more exact method of application, and we are indeed grateful to Dr. Post and his associates for devising the thermophore and providing us with exact clinical data to guide us in its use.

Personally, we have had great satisfaction in the use of the thermophore in the treatment of corneal infections, and can readily see how this instrument may give equally satisfactory results in the treatment of neoplasms when suitably located. In superficial infections and growths, a judicious use of the electrocautery doubtless would accomplish the desired result, but at best the method is inexact, and we often hesitate between the necessity for removing all of the pathology and the desire to conserve ocular tissue. This anxiety is relieved by the thermophore in handling the superficial conditions, and in addition it furnishes us with a means for the attack of lesions more deeply situated. But, as Dr. Post and his associates have previously stated, it will require a large mass of clinical data to give us the exact indications and range of this instrument.

Research studies such as Dr. Post has just given us are of the greatest value, and I trust that others may be willing to cooperate with him in determining the range and indications of this most valuable instrument in treating malignancies in and about the eye.

PARALYSIS OF CONVERGENCE.

WARREN S. REESE, M.D., F.A.C.S.

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Paralysis of convergence is a rare condition. Indeed, its occurrence as a clinical entity is doubted by some, and by others it is confused with convergence insufficiency. Thus, M. Gayet, in discussion of Grandclement's case, disagreed with the diagnosis, and considered the case one of insufficiency of convergence. He stated that a diagnosis of paralysis of convergence would presuppose the existence of a center for convergence, for which there was no proof, anatomically or pathologically. Israel, in reporting three cases, refers to this confusion. Tamagni describes paralysis of convergence as a simple concomitant strabismus with persistent diplopia. It is important that the difference between paralysis and insufficiency of convergence be understood, since in the former, the disturbance (as in all associated paralysis) is central, whereas in the latter it is peripheral.

The following case is unusual in that recovery occurred, and that within a comparatively short time:

J. W. S., male, age 26, was first seen by me on February 4, 1922. He stated that one month ago he began to see double. Previous to this, he had enjoyed good health. As a child he had had measles and mumps. In September, 1918, he was struck on the left side of the head with a 100 lb. weight, but recovered without any apparent after effects. In August, 1919, he was operated on for intestinal diverticulum and left inguinal hernia, but had an uneventful recovery. His father, three paternal uncles and three paternal aunts had all died of apoplexy, which manifested itself in each case as a left hemiplegia.

Examination of the patient showed that the ocular movements were good in all directions, but he was entirely unable to converge. The pupils were equal and active to light, but did not react to accommodation or to attempted convergence. There was no paralysis of accommodation.

On February 6, the patient was refracted under homatropin and took in the right plus 1.00 with plus .25 ax 180, and in the left plus 1.25 with plus .25 ax 180.

On February 9, he had single vision for distance, and saw double only when the finger was brought to a distance of 28

inches. The pupils now reacted to convergence at times. A postcycloplegic test was done and the following prescription given: O.D. plus .50 with plus .25 ax 180, and O.S. plus .75 with plus .25 ax 180. He had previously been wearing concave lenses.

On February 13, he had single vision at 16 inches, but stated that he saw double at a distance more easily than when he had the drops in his eyes. On this date, the Wassermann was reported plus 2, and the neurologic and medical examinations negative. At this time he was seen by Dr. William Zentmayer, who confirmed the diagnosis and suggested that the cause of the condition was probably a small central hemorrhage.

On February 16, his convergence near point was six inches with his glasses and eleven inches without them. He stated that the glasses helped the diplopia immensely.

On March 4, the diplopia had disappeared, but the patient stated that he could not do without his glasses. His convergence near point with glasses was three inches.

There are several interesting features in the above case to which I should like to call attention. The history of a right sided apoplectic attack in seven members of his father's family is unusual and suggestive. Whether this has any bearing on the present condition is of course, problematic. Another, and an equally inexplicable feature, was the fact that recovery began coincidently with the use of homatropin for refraction. There are several things which point to this being more than a coincidence, however. The patient remarked after the cycloplegic had worn off that he saw double more easily at a distance than he did while under the influence of the drops, and during recovery he could converge more with his glasses than he could without them. Holloway, in a verbal communication, recalls a case in which there was a similar improvement.

The literature on paralysis of convergence is rather meager, a large portion of it having been contributed by Parinaud. He claims to have been the first to call attention to this affection (*Arch. de Neurologie*, 1883). He distinguishes two types; first, a combined paralysis of convergence, in which there is paralysis of the muscles of elevation or depression, or of both these functions, along with paralysis of convergence. Lateral movements and movements of the eyelids and iris are not involved. The second type is an essential paralysis of convergence, in which the other movements show no tendency to change, and which has a special symptom complex, as follows: Faulty convergence, crossed diplopia in all fields without notable deviation of the

image, absence of the pupillary reflex of convergence and accommodation, and lastly there may or may not be double paralysis of accommodation without mydriasis.

The seat of the lesion in these cases has not as yet been fully determined. Parinaud suggests that it may be in the cerebellum and refers to Meniere's disease as being in some ways analogous. He also cites the experiments of Duval and Laborde as having established the influence of the cerebellum upon the coordination of movements of the eyes, especially the dissociated movements. Later on, he states that it is not impossible that the lesion, instead of directly attacking the bulbopontal nuclei, is situated in a neighboring center, which acts immediately on them, and it appears that this center may be the tubercula quadrigemina. This appears to be the result of Wernicke's observation in a case of paralysis of elevation in each eye with conservation of lateral movements, where autopsy proved a lesion of the right corpus quadrigeminum, and of another by Hensch, in which the paralysis of elevation in each eye was connected with a well defined tubercular lesion of the left posterior quadrigeminal body.

Straub reports a case following or rather complicating a third nerve palsy. He places the lesion in the oculomotor nucleus, and states that Kohler and Pick have shown that the center for accommodation and pupillary reaction is in the immediate vicinity of the center for the internal rectus.

Peters reports two cases, one of which apparently resulted from trauma of the head. There was a history of trauma in Hayne's case, but he did not consider it the causative factor.

Farnarier reports a case, following diphtheria, which did not clear up in spite of intensive treatment.

Grandclement presented a case under the caption of "Essential and Temporary Convergence Paralysis in Adolescence." He states that this usually appears about the 12th year, progresses until the 15th or 16th, and finally disappears about the 18th year. He refers to a German physiologist as having remarked that in certain animals irritation of the habenula, which is a small nucleus in the grey matter near the quadrigeminal tubercles, produces a movement of convergence.

Cestan reports a case of paralysis of convergence in which up and down movements were involved. The patient showed some hesitancy of gait, with a tendency to lean towards the left. He refers to a case of Spiller's, in which autopsy revealed a tumor in the middle of the peduncle and under the Aqueduct of Sylvius, which had caused degeneration of a large number of cells

and nuclei of the common oculomotors. Cestan states that paralysis of the up and down function and of convergence indicates a lesion of the upper part of the cerebral peduncles.

Teillas discusses these combined paralyses and refers to Priestly Smith's case of a man, aged 60, in whom the combined downward movement of convergence and accommodation was lost, while other movements were intact.

The pupillary reactions in this affection are very interesting, being (as Parinaud remarks) exactly reverse of those seen in the Argyll Robertson pupil. Straub infers from his case that contraction of the pupil is associated with accommodation, but not with convergence. The present case, and most of those reported, do not bear out this view; in fact, they seem to controvert it.

In my search through the literature, I found no experimental work bearing on this subject. Such work will undoubtedly be necessary before the mechanism of these paralyses is fully explained. Judging from the above cases, it would appear that such efforts should be directed towards the quadrigeminal bodies and the cerebellum.

BIBLIOGRAPHY.

- Peters, A. *Centralbl. f. prakt. Augenh.*, Leipz., 1889, xiii, pp. 225-229.
 Tamagni. *Ann. di Ottal.*, Pavia, 1891, xx, p. 68.
 Teillas. *Ann. d'Ocul.*, Par., 1899, cxxii, pp. 19-29.
 Grandclement. *Lyon Méd.*, 1894, lxxvii, p. 401.
 Hayne, H. W. *Arch. Ophth.*, N. Y., 1896, xxv, pp. 328-332.
 Parinaud. *Receuil d'Ophthalmol.*, 1889, xi, p. 735.
 Parinaud. *Brain*, ix, 1887, p. 330.
 Straub. *Arch. Ophth.*, N. Y., 1896, xxv, pp. 325-328.
 Israel. *Amer. Jour. of Ophth.*, Nov., 1921, p. 856.
 Cestan. *Soc. Méd. des Hôp.*, 1913, S. 3, xxxv, p. 1172.
 Grandclement. *Lyon Méd.*, 1912, cxix, p. 142.
 Farnarier, M. F. *Marseilles Méd.*, 1913, I, p. 363.

DISCUSSION.

DR. WILLIAM ZENTMAYER, Philadelphia, Pa.: Convergence paralysis is a rare condition. The case which I saw through the courtesy of Dr. Reese is the only one I remember having seen. There can be no doubt, however, that it does occur from central lesions, and that the location of the center of convergence is still in doubt.

According to Spiller, Mariana's investigations have made it doubtful whether a center for convergence in the oculomotor nucleus exists. He has shown that when another ocular muscle was transplanted and allowed to unite with the tendon of the internal rectus, contraction of the pupil in convergence still occurred, although the internal rectus muscle had no part in the convergence movement of the eyeball. Contraction of the pupil occurred when the eyeball was drawn inward through electrical irritation of the internal rectus, and even when it was drawn inward by forceps without muscular action. Duane says, that larger lesions in the pons above and below the abducens nucleus cause combined paralysis of lateral movements and convergence. Roemer states, that the tract for

the innervation of convergence separates from that running to the corpora quadrigemina, which transmits the innervation for the lateral movements. According to Knapp, the center of the coordination without parallelism (convergence) is situated in the pons, but is not exactly localized. Uhthoff saw convergence paralysis in 2 cases of occipital injury.

In reporting 2 cases, Passetti says that it is evident that there are in man centers which preside solely over convergence, these centers being situated outside the nuclei of origin of the motor nerves, and the center of association for pupillary movements. Gordon Holmes believes that a lesion of the anterior end of the midbrain will involve upward and downward movements and convergence in this order, as it progresses from before back.

Dercum states, that convergence paralysis occurs with rarity in tabes; Rochon-Duvigneaud and Heitz found it but 3 times in 76 cases.

According to Duane, it is met with in tabes, multiple sclerosis and other central nervous diseases, either functional or organic.

Bollock records a case of paralysis of convergence associated with paralysis of elevation and divergence, and of movements toward the left. The case was one of encephalitis in a child 11 years old. In discussion Ellett mentioned 2 cases due to encephalitis lethargica.

Extreme instances of convergence insufficiency are seen in hysteria. De Schweinitz says that it may be so great as to constitute a convergence paralysis; cases were observed by Parinaud, Borel, and Schweiger.

In the Möbius sign in exophthalmic goitre (hyperthyroidism), we sometimes have the same high degree of convergence weakness independent of any mechanical interference caused by extreme exophthalmus. In both these classes it is doubtful whether the term paralysis is rightly used. I have seen convergence paralysis simulated in binasal hemianopsia.

In a recent publication, *Handbuch der ärztlichen Erfahrungen im Weltkrieg, 1914-1918* (Augenheilkunde, Axenfeld), Bielschowsky says: "Concerning paralysis of convergence and divergence as focal symptoms in cerebral war injuries, there are not only few communications but their value is restricted, as in many of the cases the possibility that the symptoms were of functional origin is not excluded. The case described by Uhthoff, of hemianopsia from injury to the cerebellum, showed also more or less definite weakness, that is, paralysis of convergence.

In the literature of psychic disturbances of the eye movements, in war records, paralysis of convergence plays a relatively large part, while as a result of organic brain lesions, it was apparently seldom observed. Bielschowsky states that he has gone into the diagnosis of true organic convergence paralysis, and would say that convergence weakness, so-called insufficiency, as a functional disturbance occurs quite frequently in weakly nervous but otherwise healthy individuals. In hysteria, the frequently related cases of convergence paralysis, or weakness, is quite correctly attributed by Bartels to be an evidence of lessened will power, so characteristic of neurasthenics.

The functional can be differentiated from the organic by the presence of a considerable, if not entirely normal, convergence power. This can be determined by the use of double prisms and the haploscope. In the employment of these tests, the patient is unconscious of the use of convergence innervation.

TRANSACTIONS
OF THE
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OF THE
American
Academy of Ophthalmology
and Oto-Laryngology

OTO-LARYNGOLOGICAL DIVISION

THE TREATMENT OF NASAL SINUS DISEASE IN INFANTS AND YOUNG CHILDREN.

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IOWA CITY, IOWA.

What I have to say regarding the treatment of paranasal sinus disease in infants and young children applies to the treatment of this condition in Iowa. Our moist, changeable winter climate is a very prolific producer of paranasal sinus infections. It prevents a cure in many cases of chronic sinuitis. Certainly, paranasal sinus disease is a different thing in Iowa than in Tucson, Arizona. Cases which do not respond to treatment in Iowa improve as if by magic if the patient goes to Arizona.

In short, the thing which has the greatest bearing upon the prognosis in chronic paranasal sinus disease in infants and young children is the ability of the patient to seek a proper climate. Naturally, this is the most important thing in the therapy.

The second most important thing in the treatment of paranasal sinus disease in infants and young children is diet. Dr. Amy Daniels, the Research Dietitian in the Department of Pediatrics in the University of Iowa, has expressed her opinion regarding this matter as follows: "We have observed that animals fed diets quite lacking or very low in the so-called fat soluble vitamine ('Vitamine A') are subject to paranasal sinus infections, infections of the middle ear, a condition which precedes the eye manifestations—xerophthalmia—characteristic of animals fed diets low in this vitamine. In such animals, the addition of substances, butter fat, cod liver oil, leafy vegetables, etc., which are rich in Vitamine A, corrects the condition if dietary therapeutics are inaugurated in time." Whether or not the paranasal infections in children are infections superimposed on an undernourished condition, the result of diets low in the fat soluble vitamine, is not clear. It is significant, however, that in general, those babies receive feeding formulas low in fat, carrying from 1% to 1.2% in the total mixture, whereas those infants receiving diets carrying a fairly high percentage of available fat, from 2½ to 3%, have shown no signs of mastoid infection. When a child has a paranasal infection, the diet alone cannot correct the condition. The source of the infection must be removed first, and then proper dietary regimen instituted. Excepting for babies,

the diet should contain a fairly liberal amount of butter, vegetables, fruits, milk, eggs and cereal foods. In so far as possible, all fat given should be supplied in the form of butter or cream. Succulent vegetables, at least two servings a day, and fruits, more especially orange juice, should be used freely, since these furnish the antineuritic vitamine necessary for appetite stimulation. The amount of carbohydrate foods should be limited, for there is a tendency to the use of too much bread, potato, and cereal in the diets of most children. To make sure that a liberal amount of the fat vitamine is being supplied, cod liver oil once or twice a day is also given. *In cases of this sort it is essential to see that the diet prescribed is taken.* Children have food prejudices and frequently choose those foods, more especially the carbohydrates, to which they are accustomed.

Dr. George Smith, the Assistant Professor of Bacteriology in the Yale Medical School, working with Dr. Isabel Wason, has come to the conclusion that there is in deficiently fed animals definite evidence of a disturbance in antibody formation, that is, bactericidal substances and opsonins. They feel that these changes are indirect, being dependent upon a modified cellular activity induced by failure to provide a proper regimen.

Dr. Lambert, Professor of Pathology in the Yale Medical School, working with Dr. Judkin, came to the conclusion that the experimental xerophthalmias produced in rats fed on diet deficient in fat soluble Vitamine A are essentially inflammatory, and obviously bacterial in origin. His idea is that such animals are rendered by a faulty diet particularly susceptible to infection. He further says that it is interesting and difficult to understand why the infection should involve particularly certain tissues like the conjunctiva and, as we have found in Iowa, the paranasal sinuses. He also says there may be other infections which have been overlooked. He further calls our attention to the fact that on a different kind of deficient diet, which Dr. Underhill has employed in dogs in an effort to reproduce pellagra experimentally, a very different infection, namely, a widespread and severe stomatitis and pharyngitis, is brought about, all of which Dr. Lambert feels points to a possible relation between diet and infection.

In bringing these matters to your attention, I do not wish to give the impression, that because at Iowa it has been found that rats fed on a defective diet, one deficient in fat soluble Vitamine A, developed in every case an infection of the paranasal sinuses, and that this infection has not been found in any normal rat or

in any rat fed on a diet deficient in calcium, when experimental rickets has been produced, it is the absence of the fat soluble Vitamine A in foods which is responbile for the presence of paranasal sinus infection in infants and young children. I do feel, however, that all these observations we have cited suggest that errors in diet play a most important part in causing paranasal sinus infection in this class of cases, and correction of errors of diet is a most important therapeutic agent. I have taken a half a dozen cases of paranasal sinus disease, chronic in infants and young children from three to seven years of age, and after doing everything that we could do from the rhinologic standpoint for these patients, we have turned them over to Dr. Daniels for feeding, with a most remarkable improvement, not only in the paranasal sinus disease but in the outlook of the patient. These patients remain in our ward under the same conditions which had previously existed, and the same treatment was continued. The only difference was the change in diet.

In addition to climate and diet, proper hygienic conditions as to dress, foot wear, the living and sleeping rooms is of importance.

Certainly, in acute paranasal sinus disease in infants and young children, bed rest, diet, ventilation of the room and sunlight are the essential things in its treatment. This, with a hot nasal irrigation followed by dropping into the nose some penetrating antiseptic as argyrol or mercurochrome, plus perhaps suction treatment, is all that is usually necessary.

In every case of chronic nasal sinuitis, diseased tonsils and adenoids, if present, must be removed. In one eighteen months old baby with a posterior cleft, after the removal of adenoids the most persistent treatment for two months would not obliterate a hemolytic streptococcic sinus disease. The tonsils were diseased. It was considered inadvisable to remove these before closing the posterior cleft, because of the contraction of the pillars of the fauces. As the cleft could not be closed until the sinus disease was cured, the tonsils were finally removed. Following their removal, the nasal sinus disease rapidly disappeared, and the posterior cleft was readily closed.

Eighty per cent of our chronic cases of nasal sinus disease in infants and young children have been cured by the removal of tonsils and adenoids without any other treatment. These favorable results, I feel are due in great part to first, the age of the patient excluding long chronicity; and second, the comparative infrequency of nasal obstructive lesions in this class of cases.

While nasal obstructive lesions in infancy and early childhood are not common, they sometimes are present, and are important etiologic factors in the production of the sinus disease. In such instances, this condition must be corrected, if the work can be done without interfering with the development of the nose.

The local treatment of chronic suppuration of the paranasal sinuses in this class of cases consists of nasal irrigation, suction treatment and the use of argyrol or some similar drug. An alkaline solution as hot as can be comfortably borne should be used. The container should not be more than eighteen inches above the head, and the patient should lie on a table face downward, and with the head lowered over the end of the table. The fluid is lowered into one nostril and flows freely from the other, without coming in contact with the pharyngeal opening of the tube. The argyrol may be introduced into the nares by using an eye dropper.

There are various kinds of apparatus suitable for the suction treatment; the Brawley suction; the Lore, combining suction and nasal irrigation; the Coffin, using alternately negative and positive pressure, the positive pressure being used to force some silver preparation into the nose; the Haskin method, which is a very valuable method of using suction in infants. To use this, an ordinary catheter is attached to a Carmody suction apparatus, and the catheter is introduced into the nose and then brought near the ostium of the sinus and suction applied.

In chronic maxillary sinusitis, it is sometimes advisable to aspirate the maxillary sinuses and to inject 1% argyrol or some similar drug. This is done by passing a very small trocar through the meatal wall into the sinus. A long blunt needle, to which is attached a Luer syringe, can be inserted into the sinus through the trocar. The sinus is easily aspirated and argyrol is injected.

Rarely is it necessary to make some opening into the antro-meatal wall through which the sinus can be daily irrigated. I doubt that it is ever necessary to open the maxillary sinus by the buccal route in infants and young children. Only very rarely is it necessary to operate the ethmoidal and sphenoidal sinuses. They should be operated only when the sinusitis refuses to yield to persistent energetic treatment, and there is a severe local or systemic complication which would justify operating.

DISCUSSION.

DR. M. F. ARBUCKLE, St. Louis, Mo.: I have seen many cases of sinus disease in children. My results are, in the main, like Dr. Dean's, and my treatment is more or less along the lines he follows. The idea of sinus disease in children, especially in young children, is still scoffed at by the general practitioner and many specialists. But if the men will undertake to study these diseases and treat them, the results will be most satisfactory and thrilling at times. Dr. Dean mentioned climate, and I think that is a very important factor, particularly in convalescence. If adults who have sinus disease sufficiently severe to require operation for relief, can be operated and then go to a warm, dry climate, the prognosis as to ultimate recovery, I think, is very much better.

The treatment at the St. Louis Children's Hospital has very rarely been operative. Most of them get well without operation; but where this is required, I think it should be carried out, regardless of which sinus is involved. Occasionally, children who have recovered from their constitutional disease will return with a recurrence. I think these cases will very often require operation. I believe the results are more satisfactory in children than in adults, in that the results are obtained more quickly, and the effect on the general constitution is more satisfactory.

As far as vitamins are concerned, I have had no experience with them. It is striking, in cases of sinus suppuration in children in which there is loss of appetite, loss of weight and energy, that they will rapidly regain their appetite and come back to a more or less normal childhood, and want to play and sleep normally after these infections are cleaned up. Recently, I had a child who did not wish to play or do anything the other children did. She had a generalized nasal infection, not confined to any particular sinus. Two weeks later, the mother told me that she was about to eat them out of house and home, that she had had two dinners the previous day, whereas before they could not get her to eat at all.

For application to the mucous membrane after the sinuses have been cleansed, as outlined by Dr. Dean, mercurochrome has been most satisfactory in my hands. In the antrum, gentian violet has served me better than anything else, used in a strength of 1:2000.

Many men say there is no use in operating the sinuses of children, because they close up right away. I do not agree with them. I believe they stay open if properly operated, and that the children get well, and that if there are recurring infections, it is easy to go in and treat them.

DR. HENRY B. LEMERE, Omaha, Neb.: I think we are indebted to Dr. Dean and Dr. Daniels of the University of Iowa once more for advancing us a step further in the treatment of nasal sinus disease in children. The work of Dr. Daniels proves that dietary measures are of great value in the nasal sinus diseases of children. They are probably more effective as prophylactic measures than as a cure of the disease after it is once established. The treatment of these diseases in children, I agree with Dr. Dean, can never be expected to eradicate the disease in our climate. In many instances, we have to carry those children along until they reach their proper development, with more or less nasal sinus trouble all the time.

I wish to call attention to one method of treatment that can be used in the office, and that in my hands has been very effective in carrying these children along with good nutrition, and with no signs of the sinus disease that has been existing all the time, and that is the use of the postnasal spray. At first this seems to be a terrible thing for these children, and there is usually a struggle and fight during the first two or three applications, but after that they accept the treatment very willingly. Then the parents are provided with a DeVilbiss compressed air apparatus with the nasal spray, and are instructed to use this every day for cleansing the nose.

DR. EMIL MAYER, New York City: I would like to ask Dr. Dean, if he will not further favor us with a thorough investigation as to a possible causal factor in keeping up this diseased condition in the water that is used. I do not think that the climate is the cause of infection alone, but I do think that, in some cases, the water that is used for cleansing purposes is so hard, that it rather keeps up the disease than otherwise. I have been led to this conclusion by the results in two children in the last six months. One case, a little longer ago, was in a relative of mine in Nebraska, who still lives there. This little girl was developing sinus trouble, and was taking plunges in the big indoor tank they have in that town. In the other cases, it was the same thing. I was asked if this little relative of mine could continue her plunge baths, and I said no. If she had been near the seashore, I would have had no objection, but when this washing out of the nasal sinuses with this hard water was stopped, the nasal trouble practically disappeared.

DR. WILLIAM MITHOEFER, Cincinnati, Ohio: There are two questions I would like to ask Dr. Dean. First, how often after the use of nasal irrigations has he had an ear complication; and, second, in the animal experimentation he made, was there as much infiltration in the other mucous membranes of the body as in the nasal mucosa?

In the treatment of nasal sinus disease in infants and children, there are three points that I would like to bring to your attention. We have used the following method of procedure in all of our cases, and have had rather unusual results in most of them. First, all cases of tonsillectomies and adenoidectomies are radiographed before the operation, if they give any evidence of sinus disease. If the antra are clouded, the first step in the operation is the puncture of the antrum, not in the inferior meatus, but through the natural orifice. This is done, not with a sharp needle, but with the ordinary cannula, and air is blown into the cavity. If pus is present, a culture is taken and an autogenous vaccine is made. I believe that in these children it is absolutely essential to use an autogenous vaccine. We have had some wonderful results from their use. The second point is the use of Bier's hyperemia in the treatment of sinus disease. A rubber band is put around the neck, and allowed to remain on at least twenty hours daily. This causes an increased leucocytosis to take place, and aids in overcoming the infection. Third, we must not forget that many of these children have an endocrine disturbance which needs to be more carefully investigated.

DR. K. A. PHELPS, Minneapolis, Minnesota: There is one group

of sinus infections in children to which particular attention has not been given, and that is the group of older children where trouble occurs after the tonsils and adenoids have been removed. This requires treatment of the sinus itself. In my experience, I have found gas anesthesia excellent especially for the treatment of young children. Under this anesthesia, we can easily make our opening into the antrum. The best instrument I have found is a reverse chisel brought out by Bishop. This will permit of going in below the inferior turbinate. These cases can be opened once and irrigated once, and very frequently that is enough.

There is another type of condition that should be mentioned in this same sort of child. After removal of the tonsils and adenoids, there are often repeated nasal colds. Have they a sinus infection or not? This is what I call a "microscopic infection." The clinical examination is negative, but microscopically, bacteria are found in the washings. In these cases we treat them as if real pus were present, and very often we are able to produce a condition that does not show the repeated colds.

DR. L. W. DEAN, Iowa City, Iowa, (closing): I wish to emphasize again, if I may, the importance of removing diseased tonsils in these cases of paranasal sinus infections in infants. We had an infant with a posterior nasal cleft, with discharging ear and a paranasal sinus disease. We were afraid, that if we closed the cleft, the pus from the paranasal sinuses would infect the wound. So this child was treated for many months. We were afraid to remove the tonsils, because the resulting scar might interfere with the closure of the posterior cleft. Everything we could think of was tried without results, so far as the suppuration of paranasal sinuses and the ear was concerned. Finally we removed the tonsils, following which the suppuration in the paranasal sinuses and ear disappeared. The removal of the tonsils did not interfere with the closure of the cleft.

Eighty per cent of our chronic infections in infants and young children are cured simply by washing out the diseased sinuses, and by the removal of diseased tonsils and adenoids.

Operations upon the paranasal sinuses of infants and young children, except simple procedures for irrigation of or drainage of the sinuses, are only very rarely indicated. I have done only two external operations on the frontal sinus of children under eighteen years of age. One was a case with a serious ocular complication; the other, a case of abscess in the frontal lobe of the brain. A maxillary sinus operation by the canine fossa route is never indicated under the age of sixteen years. Operations on the ethmoids are indicated only when very serious ocular or systemic complications are present.

I quite agree with Dr. Mayer that sinus infections are sometimes prolonged by too frequent irrigation of the sinuses.

ACCESSORY NASAL SINUS INFECTION WITH COMPLICATIONS.—AN ANALYSIS OF CASES ASSOCIATED WITH ASTHMA AND NON-TUBERCULOUS CHEST LESIONS.

FRANK L. DENNIS, M.D.

COLORADO SPRINGS, COLO.

During the past ten years, attention has been repeatedly drawn to the systemic effects of chronic nasal accessory sinus disease. The fact that children may and do have involvement of the sinuses has also been emphasized. It has been demonstrated that cases of sinus disease very often exist unrecognized for long periods of time (frequently for years), because of lack of distinctive symptoms. Many of them are discovered only during routine examination when an X-ray has been taken, or an operation done, or diagnostic puncture made. Among others, my associate, Dr. Mullin,¹ has traced the association of chronic antrum disease and bronchiectasis.

In view of the above mentioned facts, the thought arises: "Is it not probable that many of these patients with disturbance remote from the sinuses, such as asthma, bronchiectasis, nephritis, arthritis, cardiac lesions etc., have been afflicted for a long time, or even since childhood, with paranasal sinus disease?"

Dean² and others have recently called attention to the role played by sinus disease in children in the causation of remote systemic conditions, particularly arthritis. This phase of the subject has only recently been given the attention its importance deserves. There can be no doubt that an unrecognized and hence, neglected, sinus infection in childhood is responsible for a great number of cases of bronchiectasis and other conditions seen in later life. The lesson is obvious, viz., greater care in examining the sinuses in children who complain of frequent colds, protracted cough with expectoration, and lassitude, and who are perhaps undernourished and anemic. In the series I shall report, almost half the cases of bronchiectasis originated within the first ten years of life, the morbidity rate falling sharply after that. One case dated the trouble from an attack of whooping cough at the age of fourteen months.

There can be no question that we are seeing many more cases of nontubercular chest disease in recent years. Perhaps this is

due to more accurate differentiation from tuberculosis, and to a recognition by internists of the relation between these cases and sinus affections.

This study embraces a series of sixty cases seen by Dr. Mullin and myself during the past two years, and includes, for the most part, cases with some remote complication. All had some sort of surgical intervention.

24 patients had bronchiectasis.

28 patients had asthma.

8 patients had other troubles; nephritis, lung abscess, cardiac involvement, arthritis.

10 patients dated their trouble from an attack of influenza.

In 11 cases, only puncture and washing of the antra were done.

In 44 cases, radical antrum operations were done, 19 of which were bilateral.

In 3 cases, the antra were opened intranasally.

In 5 cases with radical antrum operations, no counter opening under the inferior turbinate was made, the natural opening being enlarged.

In 8 cases, the ethmoids only were operated on.

In 17 cases, the sphenoid was opened, together with other sinuses.

In 1 case, the frontal sinus was opened externally.

The influence of influenza as a causal factor is shown by the great increase in the number of cases seen during the past four years, which is borne out by the experience of all doing sinus work. In the past, not a few cases of bronchiectasis have been mistaken for tuberculosis, and have even had more or less extended sanatorium care.³

A striking feature is the multiplicity of sinuses involved. In this series, only one case had trouble in only one sinus. Most of them had bilateral trouble and, in not a few, all the sinuses were involved. In practically all the cases with bronchiectasis or asthma, the maxillary sinus was diseased—47 out of 52 cases. This suggests very strongly the close relation between disease of this particular cavity and these chest lesions. It also supports the experimental work of Mullin and Ryder⁴⁻⁵ on the lymph drainage of the sinuses.

An important point, and one which I wish to emphasize, is that in many of these cases, a positive diagnosis of the condition was not possible until a direct view of the cavity was obtained. Pus in the nose, pathologic changes in the membrane of the meat-

uses, pus in the antral washings and headache were all absent. The best one could do was to label them "suspected". Mullin and the writer⁶ brought out this point in a former article, in which we argued that an exploratory antrum operation was justifiable for diagnosis. We were then discussing the advantages of the external over the intranasal operation. The argument is even stronger in the type of case under discussion here than in cases of suspected antrum disease without complications.

The X-ray as a diagnostic aid leaves something still to be desired. With our present ability to interpret the findings, we feel that it cannot be relied upon, although the writer has the conviction that, with increased experience and closer cooperation between the clinician and the roentgenologist, much more help will be had from this valuable agent. While one can say that, on the whole, X-ray findings check up fairly well with operative findings, it has happened in my experience that at times the picture has been absolutely misleading; either indicating trouble where it was not present, or being negative when at operation distinct pathology was found. This is particularly true of antrum pictures; the findings in ethmoid and sphenoid pictures were even less reliable. Thus, one case of bronchiectasis complained of discharge from the right nostril, and on examination pus was found only in this side of the nose. Two X-rays revealed a cloud in the *left* antrum. Both antra were opened. No trouble was found in the left antrum, but considerable pathologic change and pus was present in the right.

In a case of asthma of long standing, which had had numerous intranasal operations without relief and in whom all the sinuses of one side were diseased, radical operation on the maxillary, ethmoids and sphenoid has succeeded in relieving her asthma, notwithstanding the fact that the frontal sinus is still suppurating mildly. Nothing was done to the frontal beyond providing better drainage by means of the ethmoid exenteration. In nineteen cases, one or both frontal sinuses were reported by the roentgenologist as "hazy", but in only one case was it thought necessary to do an external operation.

The duration of the symptoms was from two weeks to 35 years, several patients stating that they had had trouble all their lives or "since childhood". The patient who claimed to have had trouble for only two weeks had extensive disease and pus in all the sinuses. He had been almost totally blind in both eyes for more than a year, supposedly of specific origin. So, it is certain

that his affection was much older. The Wassermann reaction was negative.

The ages of the patients varied from 6 to 66 years. The following tables show their classification by ages.

TABLE 1.

Age at examination	Bronchiectasis	Asthma
5 to 10 years	1	2
10 to 20 years	9	3
20 to 30 years	3	2
30 to 40 years	4	2
40 to 50 years	5	14
Over 50 years	2	5
	<hr/> 24	<hr/> 28

TABLE 2.

Age contracted	Bronchiectasis	Asthma
1 to 10 years	11	8
10 to 20 years	5	4
20 to 30 years	2	2
30 to 40 years	2	8
40 to 50 years	2	5
Over 50 years	1	0
Not recorded	1	1
	<hr/> 24	<hr/> 28

In the whole series, pus in the sinuses was noted 28 times, and polypoid degeneration 45 times. In the 52 cases of bronchiectasis and asthma, pus was found only 13 times; in the vast majority of them there were hyperplastic changes in the mucosa. The frequency of this latter condition and the comparatively few cases with macroscopic pus is noteworthy. In this connection, it may be stated that, owing to the minimal amount of secretion and its retention in the cavities in the hyperplastic types, the chances for and the effects of absorption are greater and are more prone to cause remote than merely local trouble.

RESULTS.

Of the 24 bronchiectatics, 16 were "improved" or "temporarily improved", 4 not improved and 5 not noted.

Of the 28 asthmatics, 19 were "improved" or "temporarily improved", 4 not improved and 5 not noted.

The immediate effect of operation on the cases of both bronchiectasis and asthma is usually symptomatically good. The

patient feels better, the attacks of asthma are ameliorated or entirely relieved, cough and sputum are lessened, and both the patient and the doctor are elated. Unfortunately, many of them gradually relapse, sometimes, no doubt, because the focus has not been entirely eliminated. However, enough cases are relieved, apparently permanently, to encourage one to continue his efforts to cure the sinus condition by operation. Even if a cure is not always possible, operation lessens absorption by providing drainage. Certainly enough are improved to demonstrate the close relation of the chest and sinus conditions. In early cases, removal of the focus of infection results, I believe, in a practical cure. This applies especially to early bronchiectasis. My impression is that asthma is not so frequently relieved permanently. On the other hand, while nothing one can do in old established bronchiectasis can correct the bronchial and peribronchial pathologic changes, we are occasionally gratified to see a long standing asthmatic restored to a life of comfort. Therefore, the conclusion is justified that these cases should be operated upon as early and as thoroughly as possible, because without such measures the outlook is indeed dark.

A few words about the method of operating may not be out of place here. Thoroughness is essential. No diseased cavity should be left undrained if relief is to be expected. This applies with particular force to the maxillaries, in which not only should adequate drainage be provided, but also complete removal of the diseased tissue from the cavity is necessary. This can be surely accomplished, I believe, only by an opening through the canine fossa with good exposure, whereby all parts of the cavity can be accurately inspected. An intranasal opening alone does not permit this, and should not be relied upon in the type of case under discussion. The reasons for this seem so strong as to not require argument. We have found that dependence upon enlargement of the natural opening for drainage is not satisfactory, and we have abandoned this procedure. One should always make a generous counter opening beneath the inferior turbinate.

Operation under local anesthesia is so satisfactory, and has so many advantages over that under ether, that it is the method of choice.

It must have occurred to many who see the extent of involvement in the sinuses, that there must be a common cause for both the head and the chest condition. Whether the sinus affection is the primary focus, or what, indeed, the ultimate cause of the trouble may be, we do not know. Perhaps the secret will be re-

vealed through blood chemistry, or the discovery of some factor of immunity (the lack of which will be proven in these cases) will solve the question. Very significant is the recently published work of Daniels, Armstrong and Hutton⁷ on the feeding of rats with a diet deficient in fat soluble A vitamine. They have shown that these animals are very prone to infection, all of them having purulent inflammatory changes in the paranasal sinuses, middle ear and eye. They suggest that this lack may be the underlying factor which determines the poor resistance of some individuals to infection. So far, however, operation offers the best means of combating the disease.

SUMMARY.

1. Infection of the sinuses, particularly the maxillary, is so common in bronchiectasis and bronchial asthma as to strongly suggest an etiologic relationship. It is imperative to investigate the sinuses in all cases of this type of chest affection.

2. Many cases unquestionably arise in early life.

3. In most of the cases, several or all the sinuses were involved, and the type of inflammation was hyperplastic rather than purulent.

4. Early recognition is essential for the best results, as little benefit can be expected from treatment of the sinuses after peribronchial changes have occurred.

5. The X-ray as a diagnostic aid cannot be relied upon in all cases.

6. Exploratory operation by means of a small opening through the canine fossa is a justifiable diagnostic procedure in suspected maxillary antrum disease.

7. Operative measures offer the best means of relief.

8. Probably a common factor is responsible for both the sinus and chest disease.

BIBLIOGRAPHY.

1. Mullin, W. V. The Accessory Sinuses as an Etiologic Factor in Bronchiectasis. *Ann. Otol., Rhinol. and Laryngol.*, 1921, v. 30, p. 686.
2. Dean, L. W. Paranasal Sinus Disease in Children. *Univ. of Iowa Studies in Medicine*, April 15, 1921, v. 2, p. 1.
3. Webb, G. B. and Gilbert, G. B. Bronchiectasis and Bronchitis Associated with Accessory Nasal Sinus Disease. *J. A. M. A.*, 1921, v. 76, p. 714.
4. Mullin, W. V. and Ryder, C. T. Studies on the Lymph Drainage of the Upper Air Passages and Accessory Sinuses. *Laryngoscope*, March, 1921, v. 31, p. 158.

5. Mullin, W. V. The Lymph Drainage of the Accessory Nasal Sinuses. *Trans. Am. Laryng., Rhinol. and Otol. Soc.*, 1919.
6. Dennis, F. L. and Mullin, W. V. Value of Direct Inspection in the Diagnosis of Chronic Maxillary Sinus Disease. *Laryngoscope*, Apr., 1922, v. 32, p. 300.
7. Daniels, Amy L., Armstrong, Marg. E., and Hutton, Mary K. Nasal Sinuitis Produced by Diets Deficient in Fat Soluble A Vitamine. *J. A. M. A.*, 1923, v. 81, p. 828.

DISCUSSION.

DR. ROBERT F. RIDPATH, Philadelphia, Pa.: I am very glad to have the opportunity of opening the discussion on this very important paper. It is only too true, as Dr. Dennis has said, that disease involving the sinuses, both of children and adults, are frequently overlooked, not only because of lack of distinctive symptoms on the part of the patient, but because of lack of knowledge of the various diseases by the physician. We must take cognizance of the fact, that sinus work really has been of few years in attaining the position it holds to-day, and not until medical colleges devote more time and attention to teaching the symptoms and treatment of these conditions will diagnosis be made. The importance of sinuitis in children, I think, has a tendency to be overemphasized. Perhaps I am making this statement because, unfortunately, I do not live in Iowa City, but we do not find so many of these cases in Philadelphia. It seems when a new condition presents itself or a new thought arises, immediately a panacea springs into being, only to take its proper place after it has withstood the test of time, and I feel it will be the same with sinuitis in children. Take our own experiences for example—Do any of you find a marked number of children affected? In Philadelphia, in the dispensary in which I work, we have fifteen to forty children presenting themselves daily, and we find very few out of each one hundred cases in which the sinus is involved. To be sure, we will once in a while find a typical, so-called "snotty nose," in which we find some involvement of the sinus, but if you will eliminate the foreign body, due to children inserting things in their own nose or in their brothers' or sisters', viz., pieces of newspapers, lemon seeds and so forth, if we remove the foreign bodies and the adenoids, I think the number will be very materially lessened. There is no question regarding the relation between lung diseases and sinus infections, as brought out by Lichtwitz in 1895, and Krause a few years later. Skillern in his fourth edition gives numerous instances and quotations.

Dr. Dennis spoke of opening the sinus and finding it healthy after roentgenologic examination. This is too frequent and, unfortunately, the roentgenologists are seemingly unable to give any explanation, and either blame Nature or us when no findings are made. Just last week, a young lady who works in a Roentgen ray studio, and has been there for four years, developed all the symptoms of frontal sinuitis except pus. The roentgenogram was negative on both frontal sinuses. A second roentgenogram was made a week later, and was also negative, and yet the patient had such severe infection that we were compelled to do an external operation. The right frontal sinus was very much involved, and large quantities of pus were discharged. We used drainage, washes

and so on, and it is unfortunate that our roentenographic examination does not give better reports.

I believe a hyperplastic condition of the ethmoid capsule with its polypoidal sequelae, decreasing the size of the nares, with its corresponding pressure causing reflex or neurosis, has more relation to asthma than suppuration. I believe that a postethmoidal or sphenoidal suppurating sinusitis, with its continuously pouring of secretion on the pharyngeal wall, gravity assisting, has more relation to bronchitis than an infection of the antra, taking the lymphatics into consideration. The almost constant coughing, trying to dislodge the tenacious mucus from the pharyngeal wall, not only brings a congestion throughout the bronchial and tracheal tree, but enlarges its lumen and reduces its expulsatory qualities, with the consequent lodgement of secretions, which again causes coughing and hacking. The local irritation of the downward flowing pus from the pharyngeal wall acts as a local excitant and irritant to the larynx.

DR. L. W. DEAN, Iowa City, Iowa: Our "follow up" records, during the last ten days, show that for eighteen months one child, six years of age, and one child, eight years of age, have been relieved from asthma by work upon chronic infected paranasal sinuses.

DR. FRANK L. DENNIS, Colorado Springs, Colorado, (closing): I do not know whether we are overemphasizing the importance of sinus disease in children or not, but so far as the cases of bronchiectasis are concerned, I do not think we are. It is so often that we find this association, particularly in young children, that I do not think we are doing our duty unless we go into the sinuses very carefully in all the cases.

One thing I want to bring out, is that so often we do not have pus. These patients have frequent colds, and may or may not have discharge from the nose, but so far as the rhinoscopic examination is concerned, we are frequently left up in the air when it comes to diagnosis.

As to opening the sinus on the roentenographic examination, I think that is a mistake, of course. The case I cited was simply to show that the radiogram is not an infallible guide to diagnosis. In that case, the sinus was clear on the diseased side, and showed an apparent infection on the other, but we thought it worth while to open the sinuses. Finding that sinus negative we simply closed it up.

THE CLINICAL AND TOPOGRAPHIC DIAGNOSIS OF SUPPURATIVE AND NONSUPPURATIVE PARANASAL CELL DISEASE.

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LOUISVILLE, KENTUCKY.

Suppuration and inflammation of the nasal accessory sinuses may be so difficult of recognition as to be almost insuperable. Pus and hyperplasia may be present or absent at the time of examination, and this variable condition calls for repeated examinations and careful routine study before a negative report can be given. In obscure cases, days and weeks may be necessary before a final report can be offered. Many examinations made at one sitting must be taken, as Hajek¹ states, "cum grano salis."

Dr. Van der Hoeve,² of Leiden, Holland, made a very significant statement at the Philadelphia meeting of this Academy in 1921, when he remarked that the rhinologist cannot say with absolute certainty that a person has no sinus affection. Many cases of latent disease of the nasal accessory sinuses are overlooked in hasty examinations. The reports of Tunis³, H. Bodkin⁴, Gavin Young⁵, Emerson⁶, Lemere⁷ and Dennis and Mullin⁸ are illuminating in calling attention to this latency. Tunis autopsied one hundred miscellaneous heads, and found macroscopic and microscopic evidence of antral disease in thirty-seven cases.

Bodkin reports his observations on the condition of the antra, based on the pathologic reports of a series of fifty consecutive mastoid operations performed upon acute and chronic cases of otorrhea. He found one or both antra infected in eighty-two per cent of the total cases; sixty-five per cent in the acute cases, and ninety-three and three-tenths per cent in the chronic cases. The number of times in which a very definite infection of the maxillary antrum was found without any symptoms to suggest its presence is rather startling, and it would have been overlooked had not a routine examination been made.

Gavin Young, in a series of thirty miscellaneous heads, found involvement in forty-three per cent of the antra and fifty-six per cent of the sphenoids. He states, that from such a small number it could not be deduced that forty-three per cent of the population of Western Scotland was affected by unsuspected antral disease and fifty-six per cent by sphenoid disease, but that

his results were significant enough to cause one to think a great deal about the latency of nasal sinus disease.

The pandemic of influenza in 1917 and 1918, followed by milder epidemics in subsequent years, coupled with the added frequency of tooth extractions, has multiplied the incidence of nasal accessory sinus disease to such an extent, that it almost seems that one of the future health problems lies in the proper sanitation of the human accessory sinuses.

With the evolution of our knowledge of the diagnosis of sinus disease, certain essential factors stand out in bold relief. In a paper of this scope, the writer can do no more than name these, emphasize and detail a few, and give common ground for discussion of the remainder.

It would be difficult to improve upon the masterful classification of the essential factors in establishing a diagnosis of sinus disease given us last year, at the Minneapolis meeting, by Canfield. In the order of their importance he enumerates them as follows: The clinical examination; the X-ray; transillumination; the history; the symptoms. He further subdivides the clinical examination in the following manner: The general appearance of the interior of the nose; the condition of the nasal mucous membrane; the presence of predisposing factors; the condition of the lateral nasal wall; the presence of discharge, its character, location and amount; the effect of change of posture; the effect of shrinking the mucous membrane; the presence of polyps; the recognition of the sinus or sinuses involved and the character of such involvement; the use of the probe, antrum, ethmoid, frontal and sphenoid; the use of suction; the bacteriologic examination; the pathologic specimen; the exploratory puncture, its uses, abuses and dangers; the condition of the nasopharynx; the condition of the tonsils and teeth.

To this outline I would add a consideration of the wiping action of the soft palate, and the condition of the membrane of the olfactory fissures, the posterior ends of the middle turbinates and the plicae septi, as seen by proper illumination. These might be subheaded under the condition of the nasopharynx, but I consider them of such importance that they should be emphasized under special headings. I would also include the use of the nasopharyngoscope.

Patients with sinus disease present themselves either as unoperated or more or less extensively operated cases, and we must attempt to determine the presence or absence of sinus disease, its character, its exact location, and its clinical importance.

We must further differentiate between a diagnosis of sinus disease, and indications for treatment or operation.

In a consideration of the practical points in the normal anatomy, topography and development of the lateral nasal wall and its associated sinuses, the writer in preparing a translation of Hajek's masterly classic "The Pathology and Therapy of the Inflammatory Diseases of the Accessory Sinuses of the Nose" has been impressed with the clear conception of the anatomy of the nasal wall to be gained from his description, in which he takes the superior maxillary bone and successively builds up the nasal hall until the large hiatus maxillaris communicating with the antrum is replaced by the small ostium maxillare. Extending upward from the large opening on the nasal surface of the superior maxillary bone is the frontal process, which is separated from the nasal surface by a groove, the sulcus nasolacrimalis, which forms the foundation for the ductus nasolacrimalis.

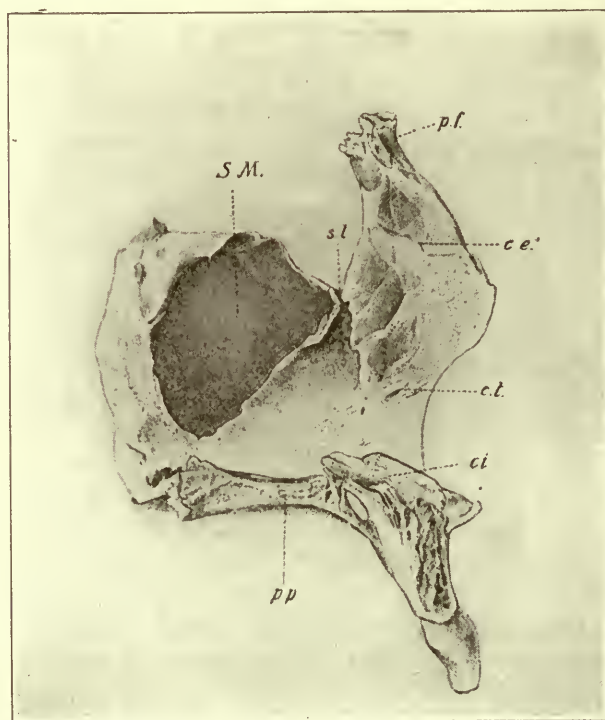


Fig. 1. The nasal surface of the left superior maxillary bone: S.M., sinus maxillaris; p.f., processus frontalis; c.e., crista ethmoidalis; p.p., processus palatinus; c.i., incisive canal; c.t., crista turbinalis; s.l., sulcus lacrimalis.

The upper edge of the large opening is formed by the edge of the orbital process, while below, the alveolar process extends downward from the antrum. The palatal process assumes a horizontal position below, forming a part of the floor of the nasal fossa. The perpendicular plate of the palate bone, by its articulation with the superior maxillary, forms a part of the nasal wall of the antrum and helps to close in the hiatus maxillaris posteriorly. The hiatus maxillaris is further reduced in size by the lower and middle turbinates, the uncinat process and the bulla ethmoidalis.

Further points of anatomic interest are the maxillary process of the inferior turbinate, which closes the lower angle of the hiatus maxillaris; the pars membranacea, which occurs between the palatal, ethmoidal, the lacrimal processes of the inferior turbinate, and several corresponding processes on the uncinat process; and lastly the hiatus semilunaris, containing in its depths

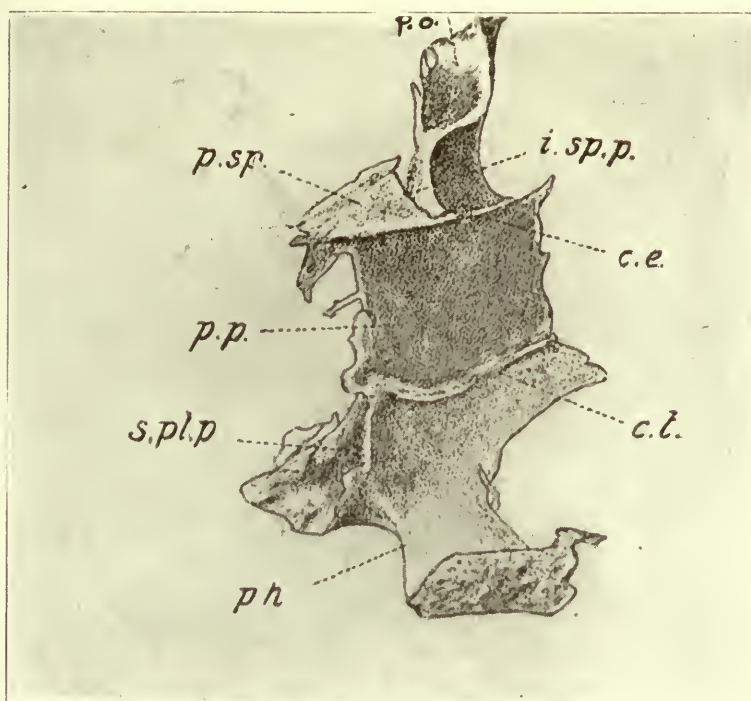


Fig. 2. The left palate bone seen from the nasal surface: p.h., processus horizontalis; p.sp., processus sphenoidalis; s.pt.p., sulcus pterygopalatinus; c.e., crista ethmoidalis; p.p., processus perpendicularis; p.o., processus orbitalis; c.t., crista turbinalis; i.sp.p., incisura spino-palatina.

the ostium maxillare. The ethmoid bone when fitted into the nasal wall completes the closure, the bulla closing in the upper angle of the hiatus maxillaris. With the duplication of nasal and antral mucous membrane bridging over the spaces existing between the processes of the uncinate and inferior turbinate, the antrum becomes shut off from the nasal fossa except in the back part of the hiatus semilunaris, where the ostium maxillare is to be found. The hiatus semilunaris is of practical importance, because into it empty the openings of the fröntal and anterior ethmoidal cells and the antrum. It will be readily seen that the difficulty in sounding the ostium maxillare does not arise from its small size, but rather in its deep lying and hidden position in the hiatus.

In conjunction with this conception of the lateral nasal wall Hajek, availing himself of the texts of Seydel⁹, Zuckerkandl¹⁰,

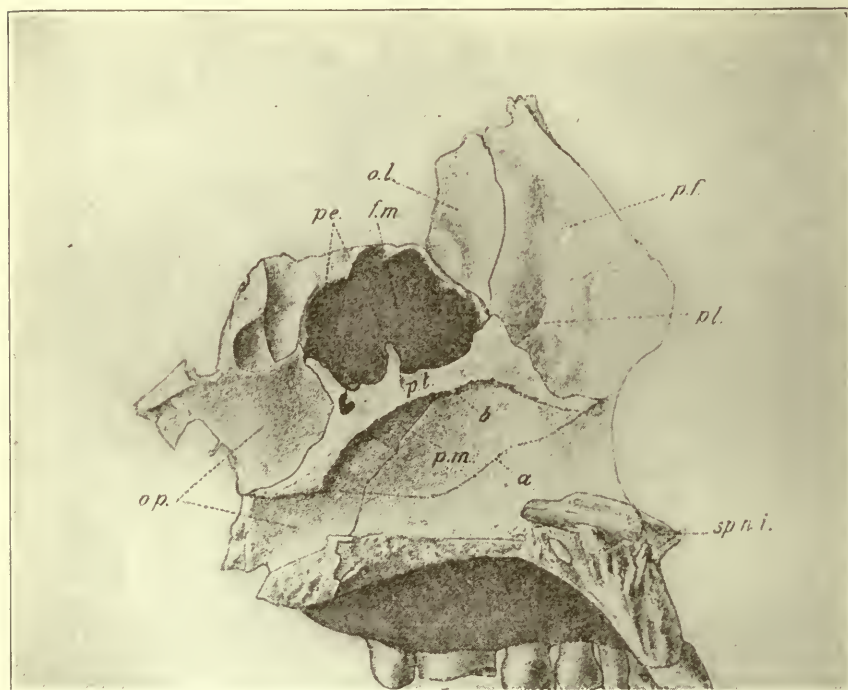


Fig. 3. The inner surface of the left superior maxillary bone after articulation of the palate bone, the inferior turbinate and the lacrimal bone: sp.n.i., spina nasalis inferior; o.l., os lacrimale; o.p., os palatinum; p.f., processus frontalis; p.t., edge of the processus turbinialis of the inferior turbinate; p.e., processus ethmoidalis of the inferior turbinate; f.m., foramen maxillare; p.m., processus maxillaris of the inferior turbinate; a., border of the inferior turbinate; p.l., processus lacrimalis of the inferior turbinate.

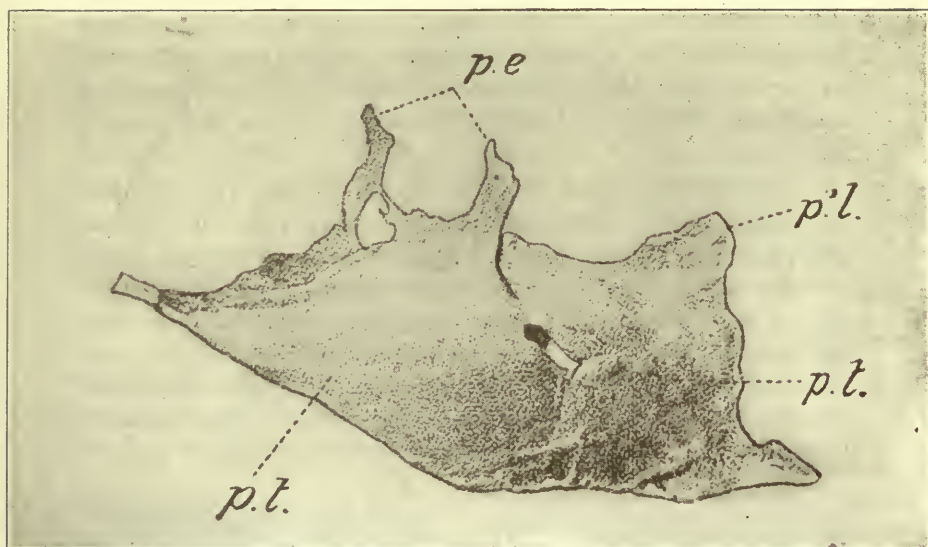


Fig. 4. The inferior turbinate of the left side seen from the nasal surface: p.t., processus turbinalis; p.l., processus lacrimalis; p.e., processus ethmoidalis.

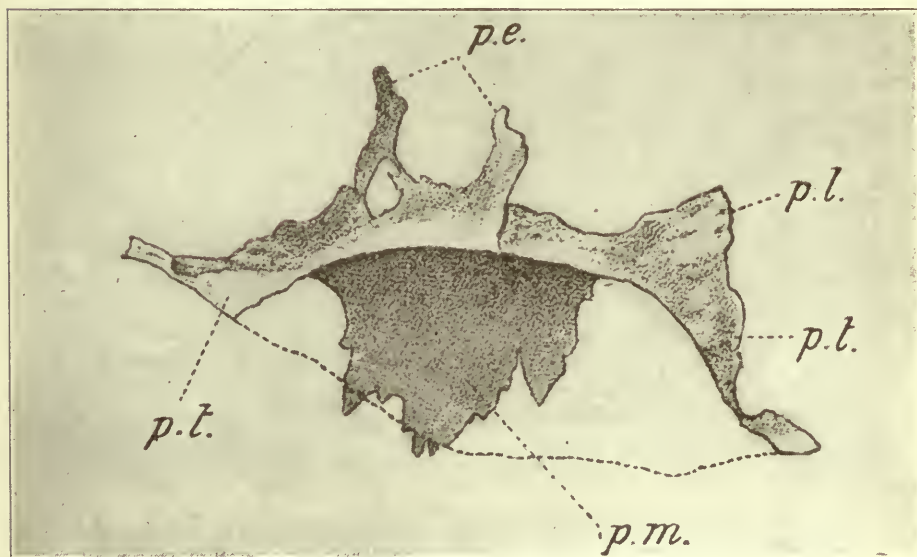


Fig. 5. The inferior turbinate of the left side seen from the nasal surface. The greater part of the processus turbinalis has been removed: p.t., processus turbinalis; p.l., processus lacrimalis; p.e., processus ethmoidalis; p.m., processus maxillaris.

Killian¹¹ and Peter¹², describes the development, architecture and topography of the ethmoid labyrinth in such a way, as to give us an excellent method for visualization of the anatomy and orientation of one's self during surgical attack on this complicated structure.

The ethmoid first develops as a number of so-called grundlamellen and interturbinalen gängen. The ends of the grundlamellen extend into the nasal fossa as the agger nasi, uncinate process, bulla, middle, superior and supreme turbinates, etc. The interturbinalen gängen appear between the lamellen and are recognized as the infundibulum, the meati, bulla cells, anterior, middle and postethmoid cells, and sphenoethmoidal recess, etc. These lamellen extend through the ethmoid outward to the lamina papyracea, upward to the lamina cribrosa and the

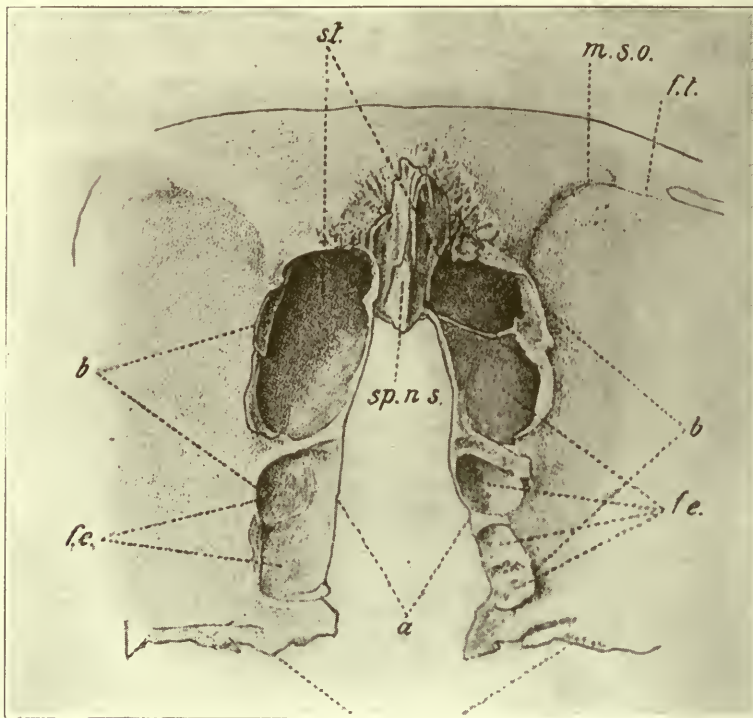


Fig. 6. Frontal bone viewed from the incisura piriformis: sp.n.s., spina nasalis superior; m.s.o., margo supraorbitalis; a., edges of the processus orbitalis which articulate with the lateral edge of the lamina cribrosa of the ethmoid bone; b., edges of the processus orbitalis which articulate with the upper edge of the lamina papyracea; f.e., foveolae ethmoidales which form the roof of the cells of the ethmoid, which are open above.

foveolae ethmoidales, which form the roof of the ethmoid, forward to the frontal bone, and inward to the inner wall of the ethmoid. The general architecture of the ethmoid labyrinth is altered by extension too far forward or backward of these lamellen themselves. These lamellen vary in number from five to seven, and in some instances there may be additional ones produced, due to a variation by a longitudinal splitting of individual lamellen.

To produce a simple visualization of this scheme, draw a horizontal line corresponding to the plane of the lamina cribrosa or foveolae ethmoidales, the roof of the ethmoid, and drop from five to seven parallel lines bent slightly to form an angle, or consider them as representing a section of an arc of a circle. The first one from before backward will correspond to the agger

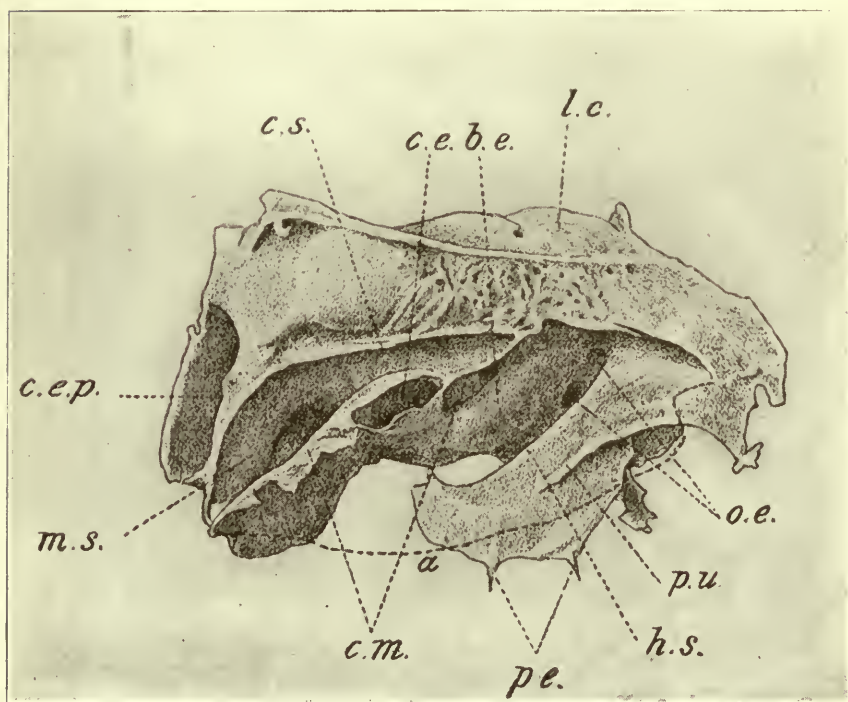


Fig. 7. The median wall of the ethmoid capsule after removal of the greater part of the middle turbinate: c.m., remaining part of the middle turbinate; a., position of the lower edge of the middle turbinate; p.u., processus uncinatus; p.e., processus ethmoidalis, processus uncinatus; b.e., bulla ethmoidalis; h.s., hiatus semilunaris; o.e., ostia ethmoidalia; c.e., cellula ethmoidalis in the middle turbinate; l.c., lamina cribrosa; c.s., superior turbinate; m.s., superior meatus; c.e.p., cellula ethmoidalis posterior.

nasi, the second to the uncinate process, the third to the bulla, the fourth to the middle turbinate, the fifth to the superior turbinate, the sixth to the supreme turbinate, etc. This arrangement gives one a general idea of the basic structure of the ethmoid labyrinth. The spaces between the lines are recognized as the interturbinalen gängen. Anomalous developments occur when the individual lamellen extend too far forward or too far backward, when the lamellen are defective or when they undergo a longitudinal splitting. The first and second lamellen are incomplete, the third, the bulla lamella, is often defective, the fourth, fifth and sixth lamellen are as a rule complete.

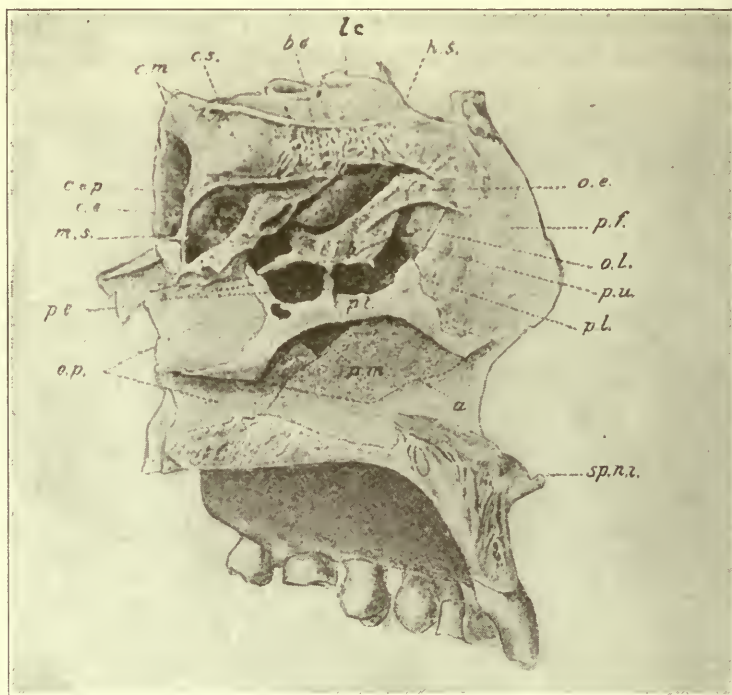


Fig. 8. The bony construction of the lateral nasal wall: p.t., processus turbinialis of the inferior turbinate, of which the greater part has been removed; a., free edge of the inferior turbinate; p.m., processus maxillaris of the inferior turbinate; o.p., os palatinum; p.l., processus lacrimalis of the inferior turbinate; p.e., processus ethmoidalis of the inferior turbinate; p.u., processus uncinatus; o.s., os lacrimale; o.e., ostium ethmoidale; h.s., hiatus semilunaris; c.m., middle turbinate, greater part removed; b., position of free edge of middle turbinate; b.e., bulla ethmoidalis; l.c., lamina cribrosa; c.s., superior turbinate; c.e., opened ethmoidal cells in the middle turbinate; m.s., superior meatus; c.e.p., cellula ethmoidalis superior; sp.n.i., spina nasalis inferior.

The ethmoidal cells are produced by the development in the interturbinal passages or gängen of septa, complete or incomplete, producing real cells or partially complete ones. The openings of the various cells determine their classification into anterior, middle or posterior cells. The anterior ethmoid is limited externally by the lamina papyracea, above by the lamina cribrosa and foveolae ethmoidales. The nasal boundaries are more variable, the main variations occurring anteriorly against the frontal sinus; in the relations of the anterior ethmoid to the hiatus and infundibulum; in the extension of cells into the middle turbinate; and in the relations of the infundibular cells. If the bulla lamella is defective above and does not reach the roof of the ethmoid capsule, the frontal sinus opens into the infundibulum and the bulla cells. If it does not reach the median wall, it fuses with the uncinate process and produces variations in bulla and infundibular cells. It may extend forward into the

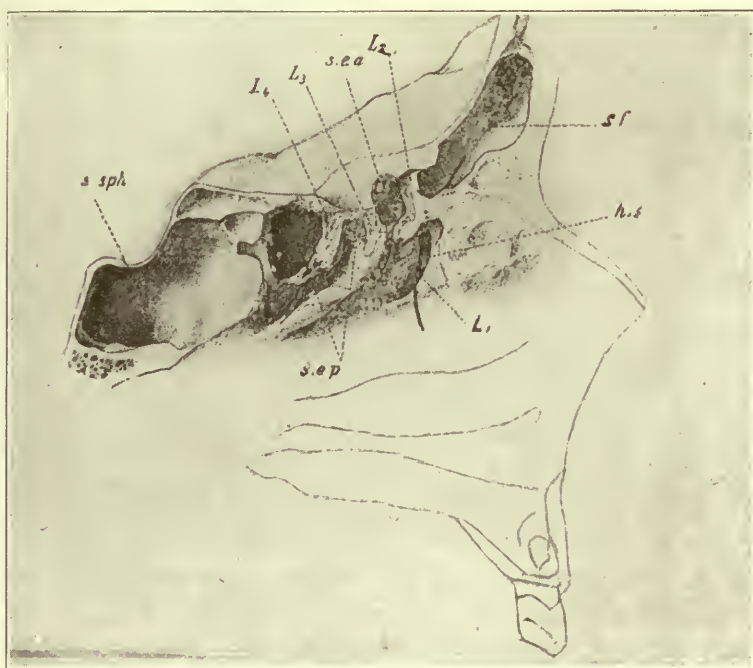


Fig. 9. The nasal surface of an adult ethmoid labyrinth with exposure of the grundlamellen; s.f., sinus frontalis; s.sph., sinus sphenoidalis; L., lamella; L₁, the processus uncinatus; L₂, the first complete lamella originating from the bulla; L₃, the grundlamella of the middle turbinate; L₄, the grundlamella of the superior turbinate; h.s., hiatus semilunaris; c.e.a., sinus ethmoidalis anterior; s.e.p., (c.e.p.), cellula ethmoidalis superior.

frontal sinus, producing the so-called "bulla frontalis." It may be situated further backward than normal, permitting an unusual development of infundibular cells to the extent that the infundibulum may be entirely surrounded by cells. A number of other types of variations of the infundibular cells occur, lack of time preventing their detailed description.

The upper portion of the fourth or middle turbinate lamella may be defective, failing to reach the roof and giving us a development of orbital ethmoidal cells, the most difficult of all to reach by any method of operation.

The main defects in the lamellen of the posterior ethmoid

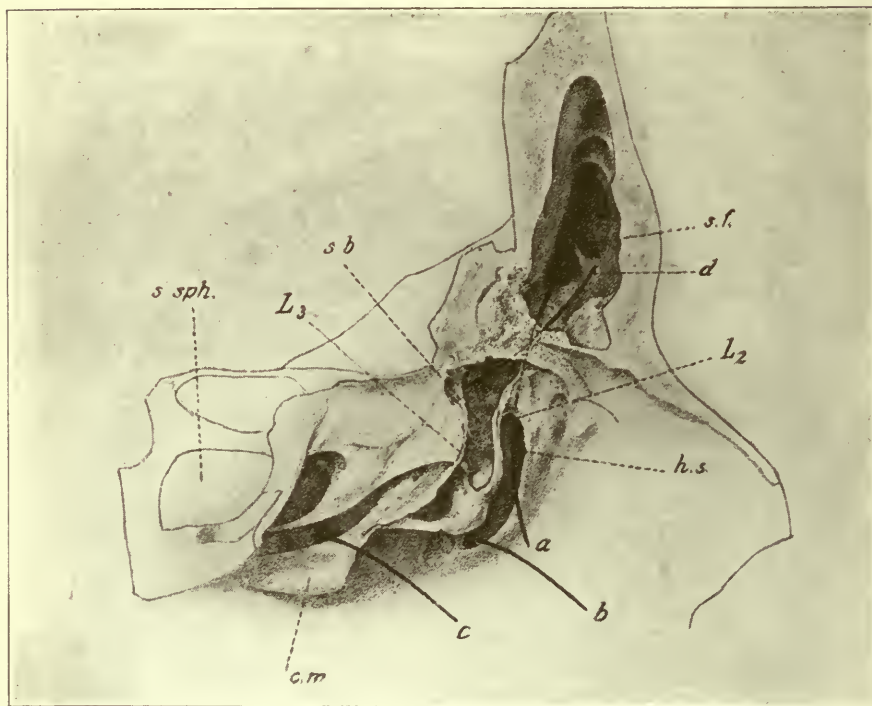


Fig. 10. The openings of a divided antrum seen from the nasal side. Anomaly of the anterior ethmoid labyrinth following a defect of the upper part of the second, so-called bulla lamella; h.s., hiatus semilunaris; L_2 , lamella 2, belonging to the bulla, is defective in that it unites with the anterior end of the uncinate process instead of reaching upward to the lamina cribrosa and outward to the lamina papyracea; s.f., sinus frontalis; s.b., sinus of the bulla, artificially opened; c.m., middle turbinate, the anterior two-thirds amputated; s.sph., sinus sphenoidalis; a., sound from the infundibulum into the frontal sinus; b., sound thrust through the ostium maxillare into the anterior portion of a double antrum; c., sound introduced through the superior meatus into the posterior part of a double antrum; d., sound leading from the frontal sinus into the bulla cavity.

occur when the ethmoid cells develop into the sphenoid, producing the so-called sphenoethmoidal cells. The upper cell is really ethmoidal, whereas the sphenoidal cell is the lower. The lamellen may fuse posteriorly, so that the anterior wall of the sphenoid forms the posterior wall of the ethmoid, or the wall of the sphenoid and the posterior wall of the ethmoid may be separated by the so-called sphenoethmoidal recess.

A thorough understanding and knowledge of this topography of the ethmoid labyrinth is important for diagnosis, orientation and treatment. It must be recognized that such knowledge is of inestimable value in making subsequent deductions as to the probable origin of pathologic secretions. Thus armed, one can with greater security and confidence carry out the dictum of Hajek—"after finding pus follow it to its source."

Topographically and anatomically Hajek divides the accessory sinuses into two series: First, those emptying into the middle meatus, including the frontal, the antrum, anterior and middle ethmoid and agger nasi cells. Second, those emptying into the olfactory fissure, including the posterior ethmoid cells and the sphenoid.

Having established the existence of purulent sinus disease, the first important question to arise is: does the pus come from the first or second series or both? If pus comes from under the middle turbinate or flows backward under this structure, we are dealing with an affection of one or more of the first series of sinuses, namely, the frontal, the anterior and middle ethmoid cells and the antrum.

Having established the source of pus in the first series, the next question which should be forthcoming is: "From which of the cells of the first series does the purulent discharge come?"

Since the opening of the antrum is at its highest point and the opening of the frontal is at its most dependent part, changes in position of the head and the effect of these changes upon the flow of the secretion may assist us. This positional test is of value only when it is positive. Experience shows that the antrum is the most frequently affected of the sinuses of the first series, and, further, that when all other methods fail, it may be washed out either through the natural opening, in a certain percentage of cases, or by a puncture through its nasal wall in the middle or inferior meatus, preferably the latter because of the danger associated with puncturing through the middle meatus.

Having cleansed the nose of pus, let us assume that washing of the antrum has given us a positive result, in that purulent

discharge was forthcoming. The question now arises, are we dealing with a frank disease of the antrum, or is it acting as a reservoir? If pus does not reappear in the middle meatus within a half hour, we may feel sure that we are dealing with an isolated disease of the antrum. On the contrary should pus reappear in the middle meatus within ten or fifteen minutes, we may rest assured that its source was not in the antrum, because the antrum could not refill in such a short time.

From whence comes this discharge? It must come from the ethmoid cells or the frontal, but which? By this time we are aware that we are dealing with a combined empyema. An effort should then be made to probe and wash the frontal sinus. Because of anatomic obstacles furnished by the middle turbinate and swollen and hypertrophied mucous membrane, a successful probing and washing of the frontal sinus will occur in only a very small percentage of cases. Further procedures being blocked, it now becomes necessary to remove the middle turbinate. When we are dealing with a combined empyema, the stage of sinus activities has assumed a complicated setting, and the middle turbinate may be looked upon as the drop curtain of the stage of sinus activities. It must be removed before the stage setting can be determined. I concur with the idea of Sluder, that a high cribriform turbinectomy should be done, in order to expose all the openings of the sinuses of the first series. This is of importance both for diagnosis and treatment. At the time of removal of the middle turbinate, it may still be impossible to probe and wash the frontal sinus with accuracy, because of bleeding and hypertrophied membrane. These will subside in a short time, when it may be determined whether the frontal sinus has been the source of the pus. If the frontal sinus has been proven "not guilty," then the pus must be followed to its source in the anterior or middle ethmoid cells.

Unless the high cribriform turbinectomy is done, the typical opening of the ethmoid cells, which lies in the angle between the bulla and the middle turbinate, will not be uncovered. With this routine procedure as outlined, it may often be a matter not only of days but weeks before an accurate diagnosis can be made. All this may be done with a minimum of surgery, namely, high cribriform removal of the middle turbinate, which is the first step in the surgical treatment of sinusitis. Frequently good surgical judgment requires that nothing more from a surgical standpoint be done.

As the exploration of the antrum is the first step in the

differential diagnosis of suppurative disease of the sinuses of the first series, the exploration of the sphenoid is the initial procedure in the differential diagnosis of suppurative disease of the sinuses of the second series, consisting of the sphenoid and post-ethmoid cells.

Having found purulent discharge in the olfactory fissure, and after eliminating surface disease, four eventualities come to mind: First, empyema of the sphenoid; second, combined empyema of the sphenoid and posterior ethmoid; third, pyosinus of the sphenoid; and fourth, empyema of the posterior ethmoid.

Soundings and washing of the sphenoid is not so satisfactory as with the same methods employed in reference to the antrum. Having determined the existence of pus in the sphenoid, the question arises as to whether the pus was secreted in the sphenoid itself, or whether the sphenoid is a reservoir for secretions of the posterior ethmoid cells. After washing out the sphenoid, if pus appears soon in the olfactory fissure the posterior ethmoids are secreting. The sphenoid opening can be packed to prevent its filling by ethmoid secretion and again washed out. If pus appears in the washings after previously cleansing the nose and removing the pack, we recognize that the sphenoid itself is secreting pus. Negative examinations are not of much value, because the posterior ethmoid cells may be empty at the time of examination. Repeated negative results are necessary to exclude the posterior ethmoid.

I have found a modified Dowling tamponade more useful than the Gottstein tamponade, as described by Hajek, in packing the ostium of the sphenoid. If the olfactory fissure is wide, the procedures as outlined can be carried out without removal of the middle turbinate; otherwise it must be removed.

In examinations of the posterior group of sinuses, one must be ever mindful of the role played by the wiping action of the soft palate, which by transferring secretion to different parts of the epipharynx may confuse the diagnosis. For the details of this action I would refer you to Sluder's¹³ reprints.

In small and medium sized pharynges, it often becomes necessary to recognize secretion anterior to the choanal plane, and here obliquely illuminated, thickened epithelium on the edges of concavities must be differentiated from purulent secretion. This differentiation becomes of paramount importance at the upper choanal arc. Proper differentiation can be accomplished only by proper illumination and repeated examination. The nasopharyngoscope may at times be employed to great advantage in clarifying

doubtful details, thereby corroborating or repudiating certain findings.

In discussing the Sluder method of the routine diagnosis of nonsuppurating nasal sinus disease, I shall confine my remarks to a few important and practical factors, referring you for further details to his monumental classic—"Headache and Eye Disorders of Nasal Origin" and his other publications.

Since the usual adjuncts in the diagnosis of suppurative sinus disease, i.e., the X-ray and transillumination, offer us so little aid in the diagnosis of nonsuppurative sinus disease, we must depend upon the chronologic history, clinical symptomatology and rhinoscopy, anterior, median and especially posterior.

Sluder has described certain minute changes in the mucous membrane and surrounding structures of the sphenoethmoidal district, visible by posterior rhinoscopy under proper illumination, which are indicative of nonsuppurative disease of the posterior group of the nasal sinuses.

Posterior rhinoscopy with proper illumination forms the sheet anchor of diagnosis in a study of nonsuppurative disease of the posterior group of nasal sinuses. Armed with a knowledge of the normal sphenoethmoidal district, one must in general translate and interpret changes in the color, thickness and translucency of the epithelium and membrane of the olfactory fissure, and the character of its vascularity, smoothness and secretion. Of further importance, variations in the size and character of the plica septi and posterior tip of the middle turbinate, which may be considered as forming the lower boundary of the sphenoethmoidal district, should be emphasized.

It is generally recognized that the best form of illumination is direct sunlight. While not always available, it must be employed with the concave mirror more or less out of focus, because when focussed the light becomes too hot for the patient's comfort. Even when used with the head mirror out of focus, there is apt to be too much glare, which is likely to obscure important details.

As the most satisfactory substitute, the arc lamp offers us a white, bright light free from a disproportionate excess of reds, greens and yellows. Lights containing greens, reds and yellows not in proper proportion interfere with a correct estimate of the true color of the mucous membrane and the thickness and opacity of its epithelium.

The forms of illumination used by most rhinologists are unsuited for satisfactory posterior rhinoscopy, both from the stand-

point of insufficient brilliance and whiteness. When compared to the arc lamp, they shine almost by darkness. A moment's comparison cannot fail to convince the most skeptical critic.

The role played by the X-ray in the diagnosis of sinus disease has not completely fulfilled our hopes, due perhaps to that human failing of expecting too much from its employment. In the past there has been too much of a tendency upon the part of the rhinologist to "pass the buck," as it were, to the radiologist, with the development of an unfair situation to all concerned, especially the patient. Expressed in the form of a Hibernicism: "It has often led to the diagnosis of sinus disease where no disease existed, resulting in operations made in good faith but with bad judgment." As a rule we can depend upon the roentgenogram to determine the presence or absence of a sinus, its shape, size and general contour, but not always the nature of its contents. The best results are to be obtained where a sympathetic relationship exists between the rhinologist and the radiologist, both able to so interpret the plates as to adjust their differences of opinion and explain their differences of observation.

The method adopted by the late Van Zwaluwenburg¹⁴ of the University Hospital at Ann Arbor, Michigan, and elaborated by Canfield at last year's meeting of this Academy in Minneapolis, accomplishes more nearly, perhaps, than any other the ideal of the radiologist, namely, the diagnosis of the character of sinus disease, suppurative, nonsuppurative, or new growth formation.

Transillumination offers us help with especial reference to the maxillary antrum. It is of problematic value in regard to the frontal sinus, and useless in so far as the ethmoid and sphenoid are concerned.

The contributions of Dean¹⁵, Byfield¹⁶, Armstrong¹⁷, Sluder¹⁸, Oppenheimer¹⁹, Killian²⁰, Haike²¹, Onodi²², Coffin²³, Emil Mayer²⁴, Smith²⁵, Mosher²⁶, Coakley²⁷ and Arbuckle²⁸ have focussed our attention upon the clinical importance of hitherto unsuspected nasal sinus disease in children. The ethmoidal cells and maxillary antra have the greatest clinical significance in infants and young children; next in importance comes the sphenoidal sinus; last the frontal.

Disease of the frontal sinus is usually excluded by the roentgenogram. Disease of the ethmoidal cells is diagnosed by anterior and posterior rhinoscopy and the use of the nasopharyngoscope.

Maxillary antrum disease is diagnosed by puncture, aspiration and culturing; sphenoidal disease by sounding, aspiration and culturing.

The X-ray is of great value in determining the presence of the various sinuses, and whether in their development they are large enough to be of clinical importance.

CONCLUSIONS.

Repeated careful routine examination and study are necessary before a negative report of paranasal sinus disease can be given.

The method of Hajek as outlined, plus posterior rhinoscopy as described by Sluder in the suppurative types, and the method of Sluder in the nonsuppurative types, offer us the best approach to a satisfactory diagnosis of paranasal sinus disease.

Paranasal sinus disease in children is often overlooked, or if considered, is dismissed as an impossibility.

BIBLIOGRAPHY.

1. Hajek. *Pathologie und Therapie der entzündlichen Erkrankungen der Nebenhöhle der Nase*. Leipzig und Wien, 1915, p. 50.
2. Van der Hoeve. *Transactions American Academy of Ophthalmology and Oto-Laryngology*, 1921.
3. Tunis. *Inflammation of the Sinus Maxillaris with Special Reference to Empyema*. *Laryngoscope*, October, 1910.
4. Bodkin, H. *The Maxillary Antrum in Mastoiditis*. *The Journal of Laryngology, Rhinology and Otology*, London, July, 1918.
5. Young, G. *The Relations of the Optic and Vidian Nerves to the Sphenoidal Sinus*. *The Journal of Laryngology and Otology*, December, 1922.
6. Emerson, F. P. *Polypoid Degeneration of the Lining of the Antrum of Highmore*. *Laryngoscope*, May, 1921.
7. Lemere, H. B. *Arch. Pediat.*, September, 1922, v. 39, p. 601.
8. Dennis, F., and Mullin, W. *Value of Direct Inspection in the Diagnosis of Chronic Maxillary Sinus Disease*. *Laryngoscope*, April, 1922.
9. Seydel. *Ueber die Nasenhöhle der höheren Säugetiere und der Menschen*. *Morphol. Jahrbuch*, Leipzig, 1891.
10. Zuckerkandl. *Zur Muschelfrage*. *Monats. f. Ohrenheilkunde*, 1897.
11. Killian, G. *Zur Anatomie menschlicher Embryonen*. *Archiv f. Laryngologie*, Bd. III.
12. Peter. *Hertwig's Handbuch der Entwicklungslehre*, 1907. Quotation from G. Kallius, *Handbuch der Anatomie des Menschen*, 13 Lieferung, 1905.
13. Sluder, G. *The Correlated Action of the Pharynx and Soft Palate, and its Effects upon Postnasal Diagnosis*. *Trans. American Laryngological Association*, 1914.
14. Van Zwaluwenburg, J. G. *The X-Ray Diagnosis of Accessory Sinusitis*. *American Journal of Roentgenology*, January, 1922.
15. Dean, L. W. *Paranasal Sinus Disease in Children*. *University of Iowa Studies in Medicine*, April 15, 1921.
16. Byfield, A. H. *Chronic Nasal Sinus Infection in Childhood from the View of the Pediatrist*. *University of Iowa Studies in Medicine*, April 15, 1921.
17. Dean, L. W., and Armstrong, M. *Some Indications for Operation on the Nasal Sinuses in Children*. *Trans. American Laryngological Association*, 1920.
18. Sluder, G. *Asthma as Nasal Reflex*. *J. A. M. A.*, August 23, 1919.
19. Oppenheimer, S. *Medical Record*, 1914.
20. Killian, G. *Zeitschrift für Ohren.*, 1908.
21. Haike. *Archiv für Laryn. und Rhin.*, 1910.
22. Onodi. *Accessory Sinuses of the Nose in Children*.
23. Coffin. *American Journal Medical Science*, February, 1905. *Trans. American Laryngological Association*, 1914.

24. Mayer, E. Medical Record, August, 1901.
25. Smith, H. Empyema of the Nasal Accessory Sinuses. New York Medical Journal, April 21, 1917.
26. Mosher. Trans. American Laryngological Association, 1914.
27. Coakley, C. Trans. American Laryngological Association, 1914.
28. Arbuckle, M. Systemic Manifestations of Suppurative Disease in Paranasal Sinuses. Journal A. M. A., September 1, 1923.

DISCUSSION.

DR. J. J. SHEA, Memphis, Tenn.: It is necessary, in making a sinus diagnosis, to consider the past experiences of the sinus under investigation. The sinuses reflect in their growth the infections they have suffered. If the sinuses are infected during the tender years of their formation, their growth may be either arrested or retarded to such an extent, that they never can fulfill their normal physiologic obligations. This will produce a persistent infantile sinus, the prototype of the persistent infantile mastoid. The stage of development of the sinuses, as shown by the roentgenogram, will influence one's consideration of the subjective and objective findings. If the sinuses are topographically infantile, their inflammations will be atrophic, while the normally developed sinuses will be hypertrophic. There is a stage which rightly can be called a cystic stage, where the roentgenogram will show the sinuses to be well ventilated and not cloudy, in fact, they may be too well ventilated and too clear. Usually the ethmoids are clarified out of proportion to the other sinuses, or else their true condition could never be appreciated. The presence of typical ethmoid cells is important, as they are prone to cause symptoms out of proportion to their pathology, i. e., an infected supra-orbital ethmoid cell will cause a severe headache, and a suprasphenoid ethmoid cell will continue to discharge after all the other points of suppuration have ceased. With the increase in the clinical phase of the sinuses, there has developed greater interest in the embryology and anatomy of the sinuses. One of the most interesting anatomic facts, is that a sinus drains into the meatus from which it originated regardless of its migration, and that the cells of one meatus do not connect with those of another group unless disease destroys their partition. This is of importance when watching a sphenoidal cavity, as the posterior ethmoid cell does not communicate with the sphenoid, and whatever comes out of a sphenoid was produced therein.

DR. W. W. CARTER, New York City: I wish to report one case that came to me a couple of weeks ago, which is very interesting.

A woman, aged thirty-three years, had influenza a year ago, followed by a right maxillary sinusitis, accompanied by a profuse purulent discharge. She recovered from this attack by the use of suction and irrigation, without operative procedure. About three months later she began to suffer with severe neuralgic pains on the right side, under the eye and along the upper jaw. A diagnosis of sinusitis was made from a general observation of the case. Two X-ray men examined the patient, and both gave a negative diagnosis. One thought there might be a bit of cloudiness over the right antrum. There was no sign of a discharge when I examined her, but she said that she could not sleep on account of the pain over the right antrum. The swelling on the right side was sufficient to be noted across the room, and this side of the face was flushed. I shrank the mucous membrane with adrenalin, but could get no discharge by suction, puncture, or any other means. I then made a puncture

of the antrum and irrigated with normal saline solution, without getting any discharge, but the woman came back the next day and said that she had had the first night's sleep that she had experienced in three months. Her relief remains up to the present time. I have repeated the irrigation once.

It occurs to me that this is a "residual antrum" with pathologic changes in the submucosa. It was a case absolutely without discharge, but responded splendidly to the irrigation of the antrum.

DR. WILLIAM MITHOEFER, Cincinnati, Ohio: The case reported by Dr. Carter brings up a point which I think is very important. I believe that before puncturing an antrum and irrigating it, the natural orifice must be probed. If this is closed, you will probably have a great deal of difficulty in irrigating. Dr. Carter probably opened the antrum with the irrigating fluid, and in that way obtained relief. I irrigate an antrum for diagnostic purposes through the middle meatus as a routine, but before introducing the cannula, I always probe the surrounding area to see if there is an accessory opening.

There is one method that has not been mentioned, and that is median rhinoscopy. This is done with a long Killian speculum, which is introduced into the middle meatus, and the middle turbinate is pushed over. In this way you can get a very good view of the ethmoid floor, and a diagnosis of hyperplastic ethmoiditis is readily made.

Dr. Heitger spoke of removing the middle turbinate. I think this should be saved. I do not believe it need be very often removed in an ethmoid operation, because in removing it, you open up the olfactory sheath and have a better chance for infection, with resulting meningitis.

DR. A. H. ANDREWS, Chicago, Ill.: In a number of cases similar to the one mentioned by Dr. Carter, in which much pain was present, it was relieved by manipulation of the antrum itself. It is my impression that the pain was due to negative pressure. The ostium maxillare had been closed by swelling. The air had been absorbed, and congestion causing much pain had resulted. These cases were relieved by puncture through the middle meatus, and on some occasions the patient and I have heard the entrance of air when the puncture was made.

I heartily endorse Dr. Mithoefer's opposition to removal of the turbinate. It is so easy to treat these cases without removal, and the end results are so much better, that I believe the turbinate should always be saved unless it is so seriously diseased in itself that it must be removed.

DR. J. D. HEITGER, Louisville, Ky. (closing): In regard to the case of Dr. Carter, one explanation was brought out by Dr. Lynch in his paper on vacuum sinusitis in the maxillary antrum. Another occurs in the work of David Thomson of London, in which, when working with gonorrheal vaccines during the war, he determined that something was happening to his vaccines. Investigation showed that gram negative organisms were dissolved in weak alkalies, whereas the gram positive organisms undergo dissolution in weak acids, such as sodium citrat.

The question of removing the middle turbinate depends upon the case.

THE MODERN TREATMENT OF OZENA (LAUTENSCHLÄGER OPERATION).

WM. MITHOEFER, M.D.

CINCINNATI, O.

It is now many years since Grünwald first insisted that the principal etiologic factor in ozena was disease of the nasal accessory sinuses, but his views have not been universally accepted. More recently, Lautenschläger has maintained that all cases of ozena are simply end results of severe infection of the nasal sinuses in childhood. These repeated infections bring about circulatory changes which affect the nutrition not only of the mucous membrane, but of the underlying bone as well. Hence, Lautenschläger believes we have always with ozena a sclerosis of the bones of the face. Thus, two factors work together to bring about the crust formation in the nose. The atrophy of the osseous framework causes a retraction of the walls, bringing about an abnormal enlargement of the nasal cavity. This, in turn, results in a disturbance in equilibrium between the nasal secretion and the nasal evaporation, and stimulates the mucous membrane to increased secretion to overcome the increased evaporation. On the other hand, the constant irritation to the mucous membrane from the underlying sinus disease causes a change in the content of the mucous secretion which becomes thicker. Thus, then, there are two factors underlying the crust formation—the widened nose and the change in quality of the secretion. It is essential, as we shall see later, to keep this two-fold condition in mind when considering the rationale of surgical treatment.

The question of why one individual with nasal sinus disease shows evidence of atrophic rhinitis with ozena and another not, is one that has not been settled. It may depend on the severity of the primary infection, or on whether or not the bones of the nose, especially of the lateral wall, are simultaneously or secondarily affected by the infection.

We have done the operation described in this paper six times, and from a study of our cases we are in a position to state, that the nasal accessory sinuses play a most important part in the etiology of ozena. In all of our patients, we found at the time of operation marked pathologic changes in the

maxillary and ethmoid sinuses. We concur entirely with Lautenschläger regarding the presence of bone sclerosis in ozena. Our patients showed extreme thickening of the facial wall of the antrum, especially in the region of the apertura pyriformis.

In considering operative correction, we must take into account the two underlying conditions mentioned above, the widening of the nasal cavity and the change in quality of the secretion. To be theoretically sound, an operative treatment should aim to check the progress of the atrophy, restore the normal secretion and narrow the nasal cavity. All operative interference such as injections of paraffin into the nose, the implantation of cartilage, the injection of autogenous blood or the implantation of buccal mucous membrane, recently proposed by Freudenthal, is but one step in the process. These procedures may prove successful in simple cases of atrophic rhinitis without ozena, but it is doubtful whether the mere narrowing of the nasal canal will ever bring about a permanent beneficial result if there is present much disease of the nasal sinuses. In fact, in all cases where an implantation of bone or cartilage into the nose has been done, several years must elapse before it can be decided how much absorption of the transplant will take place.

One of the chief aims of Lautenschläger's earlier operations for ozena, and also of the intranasal method of Halle, was the displacement of the nasoastral wall to such an extent as to cause a synechia between the inferior turbinate and the septum. We can easily understand the rationale of this displacement of the lateral wall, and should be willing to use it in our cases, but for the fact that after a certain length of time there is a tendency for the lateral wall to recede, so that after a year or two more the nasal cavity has again become wide. I appreciate the fact that, if we could maintain the nasoastral wall in close proximity to the septum, there would be a better possibility of producing a good result. Since, however, there is a persistent tendency for the wall to recede, we must look to other measures to maintain a narrowness of the nose.

Any operation to be a logical procedure should accomplish these things. It should correct the diseased sinus, thereby eliminating the irritating discharge and checking atrophy of bone and soft parts. It should narrow the nasal canal, bringing about a decrease in evaporation. Then it should stimulate

the mucous membrane to such an extent as to cause an increased flow of mucus. The Lautenschläger technic described below endeavors to fulfill all these conditions.

TECHNIC OF OPERATION

Preparatory Treatment. For several weeks before the operation, the patients should be treated daily with gauze tampons saturated with 10% ichthyol-glycerin solution. During this time, also, they should be told to drink freely of alkaline waters, preferably Vichy, so as to make them more fit subjects for operative interference. This alkalization is continued after the operation, until healing is complete. We have found that patients who have been thoroughly alkalized before operation suffer less shock and are also in less danger of a postoperative acidosis.

Anesthesia. The operation is done under local anesthesia with the following technic: Half an hour after the preliminary dose of morphin and atropin has been given, the nasal cavity is cleansed and packed with tampons soaked in 10% cocain, to which has been added eight drops of adrenalin chlorid to the dram of cocain solution. Cocain solution is also applied to the alveolar ridge, and to that part of the hard palate corresponding to the region of the posterior alveolar foramen. A 1% novocain solution with adrenalin is now injected along the alveolar ridge and along the facial wall of the antrum. About 20 drops of this solution is also injected into the region of the posterior alveolar foramen, in order to anesthetize, to some extent at least, the interior of the antrum. An important point to remember in this part of the technic is to abstain from injecting the novocain into the mucous membrane of the nose, since an abrasion of this membrane may be the cause later of an infection of the bone transplants. The patient is operated upon in a semirecumbent position and under strict asepsis.

The Incision. We use the Denker incision modified in two respects. The first difference is that the incision is much longer, extending from a little behind the malar ridge as far forward as the frenum. It is also important to incise the mucous membrane as near the alveolar ridge as possible, in order to have sufficient buccal lining for the formation of the flap which is the final step of the operation. The soft parts are gently elevated until the entire facial wall of the antrum is exposed.

Removal of Transplants. This part of the operation must be done with great care. With a straight, sharp chisel, the lines of demarcation of the two bone transplants are first made on the

facial wall of the antrum with gentle, repeated taps of the mallet. To loosen the bone from its mooring, very gentle taps of the mallet must be made at different parts along the line of demarcation. If too much force is used in chiselling, there is great danger of fracturing the bone. The transplants, when removed, should be about $\frac{3}{4}$ of an inch long and rhomboidal in shape. An assistant now takes the bone transplants, removes the diseased mucous membrane from the antral surface of the bone, and models the pieces in order to make them smooth and of suitable size for insertion into the nose.

Care of Antrum and Ethmoid Sinuses. While the assistant is preparing the transplants, the operator continues the work by carefully curetting every recess of the antrum and the ethmoid labyrinth. To do this properly is difficult enough under the most favorable circumstances. Every vestige of mucous membrane must be removed. All the recesses of the antrum must be inspected with a small laryngeal mirror. In fact, we must go even a step further and attempt to remove the superficial layer of bone of the antral wall with a hand burr or a small chisel. This treatment of the bony walls is of importance, as is shown by the microscopic findings of Lautenschläger, who demonstrated marked pathologic changes of the periosteal layer, which sent fibrous prolongations into the bone.

The ethmoid cavity is entered through the middle meatus according to the method of Jansen. Before doing this, however, we must infract the nasoantral wall in the region of the middle meatus with a very large and heavy periosteal elevator, and push it as far as possible towards the septum. When this is done, a better view of the ethmoid region is obtained. A complete ethmoid operation is now done, and the sphenoid cavity dealt with, if found diseased. The atrophied middle turbinate is infracted towards the septum. The exenteration of the ethmoid should be done through the antrum opening, avoiding intranasal work as much as possible to avoid injury to the nasal mucosa.

A small flap is now made on the lateral wall of the nose, in the region of the uncinate process. The portion of the bone underlying the flap is removed, and the flap allowed to fall into the antrum. This prevents the closure of the middle meatus opening and insures future aeration of the cavity. We are more than convinced every day that, in order to prevent all openings made into nasal sinuses from closing, a mucous membrane flap must cover the exposed bone.

Insertion of Bone Transplants. The nasal mucous membrane

on the floor and lateral wall of the nose, as far up as the inferior turbinate, is dissected with a slightly curved elevator, beginning at the apertura pyriformis. If there is much recession of the inferior turbinate, a stout periosteal elevator is placed on its under surface and the turbinate displaced upward. Care must be taken not to injure the mucous membrane of the lateral wall or the floor of the nose. If the thickness of the ascending process in the region of the apertura pyriformis prevents the easy insertion of the bone transplants, as often happens, a portion of the bone at the apertura must be chiselled away. This usually gives sufficient room for the transplants, so that one can be placed on the floor of the nose, and the other on the lateral wall under the inferior turbinate. The transplant is placed in the nose with the outer anterior wall of the bone against the mucous membrane.

Buccal Membrane Flap. The chief object of this flap is to cover the apertura pyriformis in such a way as to prevent an infection of the pocket containing the transplants. The flap is tongue shaped in contour, with the base occupying the region of the apertura pyriformis and the remaining portion placed in the antrum. The cavity of the antrum is then cleansed and tightly packed with iodoform gauze.

After Treatment. The packing remains in place for six days, and after that is changed every second day for fourteen to thirty days, depending on the rapidity of the epithelial growth in the antrum. After the packing has been finally removed, a secondary suture closes the cavity. Little is done to the nose itself. When necessary, tampons saturated with hypertonic salt solution are placed in the region of the middle meatus. It is interesting, however, to observe the condition of the nasal mucosa during the after treatment. What was once a pale atrophic membrane, within a few days assumes a congested, succulent appearance. It is to be expected that the reaction after this operation is greater than after the ordinary radical antrum. It is not as severe, however, as one might expect. Most of our patients have been fairly comfortable after the operation.

The operation is not easy of accomplishment, and presents many difficulties even under the most favorable circumstances. The results, however, have been more than satisfactory. The intranasal operation of Halle is much less difficult technically, but it is our opinion that this operation does not remove all diseased tissue in the recesses of the antrum, and that there is tendency later on, in the course of a year or two, for the lateral wall in

the region of the inferior turbinate to recede slowly. The same objection may be made to the absorption of the bone transplants, which no doubt partially occurs in the course of time. The point in favor of the transplants is the fact, that the presence of the bone transplants causes an irritation of the surrounding tissues, acting much like a foreign body, and in this way there is a tendency to hyperplasia of the mucous membrane at the site of the transplants.

In one patient, we did a Halle operation on the left side and a Lautenschläger on the right. The result from the Lautenschläger method was much better than that from the Halle procedure, so that eight months later we transplanted bone from the facial wall of the antrum into the nose, and curetted more carefully the recesses of the antrum on the side where the Halle operation had been previously done.

We believe that the technic of the Lautenschläger operation may be improved with further study of ozena, and that the number of our cases is too small to arrive at definite conclusions. Nevertheless, we know we have taken a step forward in the right direction, and that future means for the relief of this horrible malady must be of an operative nature.

Experience, only, will teach us which kind of transplant is suitable in these cases. It is true a bone transplant has a tendency to absorb, but we have argued that, since the antrum in this operation must be attacked radically, it would be very impracticable not to use the bone from the facial antral wall for transplantation. The objection to the bone transplant taken from the antral wall is the fact, that it is covered on one side by an unhealthy mucous membrane which must be carefully removed before being placed in the nose. The use of cartilage as employed by Beck and Pollock should receive some consideration, for the reason that we know cartilage does not absorb as quickly as bone. It has been our intention in some of our future cases to employ cartilage, but instead of using septal cartilage, a small strip of the cartilage from the ear, near its attachment to the bone, is to be used. One thing is certain; it makes no difference what sort of transplant one uses, the resulting irritation and circulatory disturbances are sufficient in many cases to bring about a satisfactory narrowing of the nose. This question of the use of transplants in the nose has not the ring of finality in it. We must, therefore, continue our studies until some definite conclusion has been reached.

Prolonged packing of the antrum undoubtedly increases the

circulation of blood in the nasal cavity. Rhinoscopic examination during the stage of antrum packing reveals an intumescent inferior turbinate, with the lateral wall also edematous. The intense swelling of the nasal mucosa ceases when the antrum pack is discontinued, but there persists a sufficient change of the appearance of the membrane to make us feel that it is essential to use an antrum pack, although in the last two operations we have not continued it after two weeks.

We have never attempted the technic of Wittmaack and Lautenschläger, in which they transplant a portion of the buccal mucous membrane containing Stenson's duct into the antrum in order to insure moistness of the nasal cavity. The objection to this is the flow of saliva from the nose during meals, and the danger of infection of the parotid gland. The transplanting of a portion of the tibia, or the implantation of fat into a pocket of mucous membrane of the septum, as practiced by Brunings, will not bring about the desired results if we are dealing with a true case of ozena complicated by a nasal sinus disease.

All our patients have manifested a cheerful willingness to submit to operative measures. It is necessary, however, for the operator to warn his patients, that even though the disagreeable symptom of fetor may disappear entirely or be greatly relieved, there are other symptoms which may persist after the operation and require treatment, as for instance, a pharyngitis sicca. Our cases were all true ozena, not simple atrophic rhinitis.

What were the end results? An examination made several months after the operation revealed the following important changes. First of all the fetor had entirely disappeared. The patients themselves, not having had a sense of smell, could not appreciate this change, so that the only means we had of eliciting this happy result was through personal contact and the favorable reports from members of the family. The next most striking change was the appearance of the nasal mucous membrane, which instead of having its former atrophic appearance, now assumed a hypertrophic condition. With the exception of an occasional small crust in the region of the middle meatus, this symptom entirely disappeared. There was a marked change in the mental condition of the patients, and in several cases there was a steady gain in weight after the operation.

The good results obtained in our cases may be partly due

to the fact that all of our patient had an active involvement of the nasal sinuses. According to Lautenschläger, these are the most satisfactory cases. In those patients where the sinus disease is of such long standing as to have developed into a sclerosis of the lining membrane of the sinus instead of a hyperplasia, the prognosis is said to be less favorable.

The question may arise; "Is atrophic rhinitis with ozena ever permanently cured? Furthermore, do we eradicate the disease even though we can demonstrate clinically the absence of fetor and crusts?" If we believe with Lautenschläger that ozena is a sclerosis of the bone and mucous membrane of the nose, then we must draw a reasonable inference that the sclerosis still remains, although the clinical symptoms have been controlled. Suffice it for us to say, that as a result of long application and the earnest work of Lautenschläger, we at last have a means of treating this distressing malady in a more definite and scientific way.

DISCUSSION.

DR. HARRY L. POLLOCK, Chicago, Illinois: We have had a great deal of experience with ozena and atrophic rhinitis, and while I have never attempted the Lautenschläger operation, I have done the Halle just recently. It is too early to give any definite report, but I will say that if the Lautenschläger operation is carried out by Dr. Mithoefer as I understood, he goes the reverse way from mine. In a recent paper before the Triological Society, I pointed out that the first step in the treatment is the care of the accessory nasal sinuses. That is cleared up before we attempt to do anything for the ozena itself. I earnestly believe that every case of atrophic rhinitis is associated with an ethmoid infection. I think a great many are associated with antrum infection, but not all. After the sinuses are all cleaned out, the patient is always better, but of course this does not lessen the space, and the thing is to attempt to close this huge space, especially down at the floor. In most of the cases the middle turbinate is normal, but the inferior meatus is so large that you can drive a horse and wagon through the space.

As you know, I have done a good deal of work on the septal implant, the first about four years ago. This was in a case with a deflection of the septum to one side, so that there was little secretion on that side, but there was a large space on the other. I implanted this space with cartilage and bone, and that man is still absolutely free from his ozena. In some cases I have seen partial absorption of the implant, so that it has been necessary in some instances to reoperate and put in more bone. After I had written my paper over a year ago, I tried some implants of costal cartilage, dissected up the mucopericondrium and left them in, closed with collodion, and they remained in very well. I have also taken implants from the tibia, and the result has been satisfactory. There is always some secretion, but the fetor and crust formation in all these cases have disappeared. I think I have operated on about fifty cases by

means of the septal implant. While in Kansas City, Dr. Lorie showed me the instruments he used in the Halle operation and loaned them to me. On my return home, I attempted to do what I thought was a Halle operation, but evidently I did not remove enough bone. I got a synechia. I think it was in April that I did the operation, and since then the girl has been free from the ozena.

We called attention years ago to the fact, that in cases of atrophic rhinitis the blood contained twice as much fibrin content as a normal person's. We have examined microscopically and found this large fibrin content, which I believe is the cause of the crusting.

DR. WILFRID HAUGHEY, Battle Creek, Michigan: In treating this condition of atrophic rhinitis or ozena, whichever you have, the principal thing as I see it is to give your patient relief. The Doctor gave us a very clear pathologic description of the nose and the cause of the crust formation. There is an enormous amount of space. I figure that with this enormous amount of space, the air as it passes through goes in a blast. You have a regular draft, just as through an open window, and that will dry up the secretion and produce the crusting. For something over a year, I have been treating this condition in this way: I fluff up a pledget of cotton and place it in the antrum. The patients dislike this for the first day or two, but after they wear this pledget day and night, they find it of great advantage. Some have gone for over a year, and the mucous membrane is pink and clear and the patients are comfortable. They tell me that if they leave the pledget of cotton out for twenty-four hours, the nose begins to get dry and they have crust formation. Upon putting it back, they are again comfortable.

DR. H. B. LEMERE, Omaha, Neb.: I believe that we are a little inclined to let cases of ozena go by default, because the operations described by Lautenschläger and Halle offer a great many technical difficulties. We have, however, in our own American rhinology an operation for the cure of ozena, which was advanced by Dr. Coffin in 1916, when he exhibited six cases that he had cured. These were cases of ozena taken consecutively, not particularly because they showed antrum infection, but in every case the ozena was symptomatically cured by opening up the antrum as the primary cause of the disease. I have followed this procedure in cases of ozena, and have had equally encouraging results. At times, during a cold or acute exacerbation of nasal infection, there will be some discharge and some slight odor, but on cleansing the antrum during these attacks, the symptoms disappear as soon as the acute infection has subsided. I feel that we should give credit to Dr. Coffin for the work he did in calling attention to the role the antrum plays in these cases.

DR. A. LORIE, Kansas City, Missouri: In Germany at present, there are two schools in regard to atrophic rhinitis, one following the technic of Lautenschläger, the other of Halle. Halle's is really a modification of the Lautenschläger operation. He has operated over 500 cases by this method. It has been his experience, and mine in the short time that I have been doing this work, in agreement with Dr. Pollock and not with Dr. Mithoefer, that a certain percentage do not have a pathologic involvement of the antrum. The great objection to the Lautenschläger operation is this: It is a major operation of the largest type. If the original technic is followed, Stenson's duct is drained into the antrum.

Dr. Mithoefer does not do this, but if this is done, when the patients eat they very frequently have a secretion of saliva in their nose. Secondly, if the Halle operation is properly performed, it can be finished in fifteen or twenty minutes, and there is no danger to the patient. If the results are not good, Halle's plan is not followed. The great thing is that the infraction of the ethmoid is not complete. If this is complete, the retraction of the lateral nasal wall rarely ever takes place. The Halle operation gives all the results of the Lautenschläger without any of the dangers. The odor and fetor disappear, and the discharge disappears some time later. If necessary, the operation can be repeated in the office in a very short time. Another thing in the Halle operation is that improper packing is so frequently used. The packing in the Halle operation is just as important as the operation itself. It must take place so that the upper angle of the lateral nasal wall is kept over against the septum. If this is done, you will find that the Halle operation will be almost universally successful. The operation with the local anesthesia and all should not consume more than thirty minutes.

DR. GEORGE W. MACKENZIE, Philadelphia, Pa.: Dr. Mithoefer mentioned the widened nasal space and sinus disease in the etiology of crusting, and I wish to add that there is invariably metaplasia of the epithelium in primary atrophy. Normally the epithelia have cilia, which wave toward the nasal orifice. In the case of metaplasia, the cilia are lost and the epithelium takes on more the character of flat or pavement epithelium. When the nasal discharge comes to the place where there is no cilia, it remains and crusts. All that can be hoped for by any operation is a physical narrowing of the nasal cavity, offering a better opportunity for the patient to blow his nose.

DR. WILLIAM MITHOEFER, Cincinnati, Ohio (closing): Regarding the remarks made by Dr. Pollock, I wish to say that I do not believe true ozena with atrophic rhinitis is of such frequent occurrence. We have had many cases of unilateral atrophic rhinitis without ozena, that were greatly benefited by correcting a deviation of the septum.

I have used the Coffin technic in many cases, with rather unsatisfactory results. By thoroughly removing all disease of the antrum and narrowing the clean chambers, a better result is obtained. I believe the use of cotton tampons in the anterior nares to be very good for conservative treatment, but would not consider them in the discussion of this paper.

Regarding the Halle operation: The consensus of opinion in Europe to-day is that the nasosinusal wall recedes about a year after operation and tends to occupy its former position. In the earlier operations of Lautenschläger, the nasosinusal wall was fractured towards the septum, but he now uses bone transplants instead. I have seen some very good results from the Halle operation, and some were not so satisfactory. It is true the Lautenschläger operation is more radical, but in order to insure good results every vestige of pathology must be removed from the antrum and ethmoid sinuses. This cannot be done through an intranasal opening.

LINGUAL QUINSY.

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The general appearance and anatomic character of the lingual tonsil is quite well known, but the pathologic conditions affecting it are often overlooked, not only because of their rarity, but also because careful inspection of the lingual tonsil and the possibility that trouble may arise from it are often quite completely ignored in the examination of the patient.

The lingual tonsil is separated from the muscular structure which composes the body of the tongue by only a thin layer of tissue; it is not surrounded by a definite capsule, nor separated from the tongue by a strong fascia corresponding to that of the lateral walls of the throat. Its blood supply is particularly abundant, so that when it is infected, there is a greater opportunity for blood stream infection than when an infection is located in the faucial tonsil, which has a capsule on its posterior side and is separated from the fascial wall of the pharynx by a layer of loose connective tissue. This anatomic peculiarity also makes any surgical procedure in this area somewhat embarrassing at times, on account of the danger of serious hemorrhage and the difficulty of controlling it.

The lymphatics of the tongue are very abundant and complicated, and are important in guiding the line of drainage. In lingual quinsy, the group of lymphatics at the base of the tongue behind the circumvallate papillae are chiefly concerned. These lymphatics pass to the tonsillar region and to the deep chain of glands which run along the internal jugular vein. Lymphatic communication with the rest of the tongue is not so free, although in severe inflammatory affections of the base of the tongue, the glands at the angles of the jaws, and the submaxillary and other groups beneath the tongue become enlarged.

Anatomically, the lingual tonsil is made up of an aggregate of follicles, which may be subject to infections similar in character to those which occur in the faucial tonsils. Usually, any acute infection of the lingual tonsil is a manifestation of a general infection of the lymphoid tissues in the throat, but in certain cases, such as that reported here, the infection seems to originate in and remain limited to the lingual tonsil itself.

Any acute inflammatory infection at the base of the tongue is peculiarly serious, for the following reasons:

1. On account of its contiguity to the epiglottis, there always exists a periepiglottitis, which may produce an edema and very seriously obstruct the respiration.

2. Nutrition is much interfered with because of the extreme pain caused by any movement of the tongue, and the difficulty in swallowing is much greater than when the pharynx itself is involved.

3. Because of the abundant blood supply, there is great risk that blood stream infection will result from the entrance into the circulation of bacteria from the abscess or from the breaking down of the tissue in this region.

Nearly 80 years ago, Dr. David Craigie gave the name of lingual quinsy to abscesses contiguous to or originating in the lingual tonsil. Dr. Craigie described four cases of this disease: one of these patients died, and the other three recovered only after they had been exceedingly ill for a long time. There have been very few references to this disease in the literature, and although I have made frequent inquiries among men interested in oto-laryngology, I have encountered only one who has had personal experience with a case similar to that reported below. Dr. Thompson of Manchester, England, recently gave me a report of an acute infection of this character, which occurred in one of his colleagues during the last year. In this case, the abscess fortunately pointed early, and he was able to evacuate it by incision and drainage, with a successful result.

My own case was in a shipping clerk, 31 years of age. His general health had always been good with the exception of periodical attacks of tonsillitis, and three attacks of quinsy. He was seen first on the 11th of August, 1921, at which time he had a quinsy on the left side. It was recommended at this time that his tonsils be removed. He said that he had received that advice many times, but thought that he was getting on pretty well with his tonsils. He was next seen on the 26th of February, 1923, when he appeared with a well developed peritonsillar abscess, which was evacuated with a prompt recovery. At this time, he was again urged to have a tonsillectomy, and agreed to do so when he could arrange a convenient time.

On March 26, 1923, he came in complaining of great pain at the base of the tongue and low down in the throat, accompanied by great difficulty in swallowing and some difficulty in opening his mouth. His temperature was 102 degrees and his

skin was moist and clammy. He was pale and apparently suffering. I suspected a recurrence of his tonsillitis and quinsy, but he stated that his symptoms were not like those of previous attacks. When an attempt to examine the throat was made, an enormous swelling of the tongue was noticed, which made it difficult for the patient to open his mouth wide enough to give a view of the throat. It was possible, however, to get a view of the faucial tonsils, neither of which was found to be inflamed, and there was no swelling of the soft palate. Pressure upon the tongue by the tongue depressor was painful, and the laryngeal mirror showed a very acutely inflamed condition of the lingual tonsil area, but with no pointing of an abscess. Only a small part of the epiglottis could be seen, but it was evident that there was a marked periepiglottitis with considerable dyspnea. At this time his blood count showed—leucocytes, 6500; red cells, 3,350,000; hemoglobin 75 per cent. The patient was urged to go to the hospital at once but refused; he was, therefore, told what to do for himself and asked to report again the following day.

On the following day he was very weak; his temperature was 104 degrees, and the swelling of the tongue had increased so that it was quite impossible for him to open his jaws sufficiently to permit an examination. The swelling had begun to extend to the angles of the jaws and down the sides of the neck. The patient was sent at once to the hospital, and an attempt was made to drain the abscess over the peritonsillar area to the left of the center. A small amount of very foul pus was evacuated, but apparently this drainage did not reach the main abscess at the base of the tongue. The following morning, on account of the markedly increased swelling on both sides of the jaws and the sides of the neck, it was thought best to get external drainage as well. Accordingly, incisions were made and a considerable amount of serum was evacuated. The wounds were left wide open and packed with iodine gauze. During the next twenty-four hours, there was profuse drainage of pus of offensive odor, and a greenish slough was seen at the bottom of both incisions. During the day, the patient had a profuse secondary hemorrhage from sloughing of the walls of the blood vessels in this area. He was transfused and seemed somewhat better. A blood culture at this time showed a blood stream infection of the streptococcus viridans type. By this time, there was marked diminution in the thickness and swelling of the tongue. The patient could open his mouth more easily and could swallow somewhat better. The

temperature, however, continued to be of the septic type. Within the succeeding twenty-four hours, he had two further hemorrhages, and a second transfusion was given. During one period of twenty-four hours, there was almost complete suppression of urine, but this was overcome and the daily output continued to rise. The patient continued to grow weaker in spite of the transfusions, and finally died from exhaustion seven days after his admission to the hospital. Fortunately, we were able to get a pathologic specimen, which showed the presence of an abscess at the base of the tongue of approximately the size of a large English walnut, lined with a greenish sloughing wall, and definitely connected with the base of the lingual tonsil on the left side.

As has been stated by Butlin, suppuration and abscess are caused by or complicate many affections of the tongue, such as injuries and foreign bodies; acute parenchymatous glossitis; acute superficial glossitis; caseation of tuberculous and actinomycotic nodules; the breaking down of syphilitic nodules and gummata; suppuration accompanying ranulas and calculi; suppuration of cysts—dermoid, thyroid, etc. In most of these cases, the abscesses are small, are subacute in character and are located at the sides or the anterior part of the tongue; rarely do they originate in the circumvallate papillae.

Lingual quinsy, therefore, should always be regarded as much more serious than ordinary peritonsillar abscesses, because of the danger of the extension of infection into the base of the tongue with its copious blood supply, and the greater danger of hemorrhage in an area where hemorrhage is very hard to control. I believe that a blind stab wound of any kind into the area at the base of the tongue is attended with great risk. In the particular case reported above, I think it was a mistake to attempt any form of external drainage. The best procedure would seem to be to bring the tongue forward as far as possible, under light anesthesia, and to make an incision into the area with a cautery knife.

In all cases, hospitalization, blood grouping and blood culture are desirable, and every possible supporting measure should be adopted. Preparations for emergency tracheotomy should be made and kept in readiness throughout the siege. The patient should lie on the face if possible, so that drainage may be directed away from the larynx during sleep.

DISCUSSION.

DR. EMIL MAYER, New York City: We are much indebted to Dr. Waugh for calling our attention to this remarkably unusual and very rare case. In a tremendously large clinical experience among people who might easily be subject to conditions of this kind, I do not recall ever seeing a condition so marked as he has shown here. The abscess condition has been limited to a small area. These cases are certainly more common, but are missed, in that the condition which was described so many years ago under the name of *angina ludwigii*—Ludwig's angina—is included with them. In this case, the question is whether this condition did not originate in the tonsil, for is it not possible, that the man having had a paratonsillar abscess only four weeks previous to the time he developed this abscess condition, the preceding abscess burrowed down and extended into the lingual tonsil and infected that? Be that as it may, the important thing to us is to localize these anginas in the particular space they may spring from. Is it possible, then, when we have a condition of this kind, to treat it in the manner the author suggests he would like to at some future time? Remembering that you have an abscess condition swelling up to the roof of the mouth, so that you can barely see what you are doing, can you reach this with the galvanocautery with a reasonable assurance that you have reached the right place? Remembering, too, that under general anesthesia you have to press down very violently in order to see where you are, I think you are very likely to break that abscess in a most unfortunate position, in that the patient may be turned in such position that the pus may rapidly enter his lung. I would prefer, by far, the external route in order to get at the abscess. The condition of this particular patient, with a blood count showing that he was in an intensely serious condition, made it very unfortunate that the man would not obey the Doctor's suggestion of going into the hospital. I think we should impress upon our patients the great gravity of their condition, and then if they refuse proper care, the result is on their own shoulders.

I am greatly indebted to Dr. Waugh for bringing this matter before us, and am sure it will lead to closer observation of these thick swellings which occur and have cost so many valuable lives. I believe history shows that the death of George Washington was due to an infection of this kind.

DR. MARGARET F. BUTLER, Philadelphia, Pa.: About two months ago, I had an emergency call early in the morning to see a patient, who was said to have been choking and suffering a great deal during the night. I advised him to go to a hospital, and when I saw him, I inferred from his speech that he had a peritonsillar abscess. He gave a history of some swelling with more or less pain during the preceding two weeks. I found the fauces free of pathologic disturbance, with the exception of redness and swelling of the left lateral fold. There was great difficulty in opening the mouth, and much tenderness on the left side, at the angle of the jaw. On examination with the laryngeal mirror, I found a red, glistening mass at the base of the tongue on the left side. On palpation, this was tense and about the size of an English walnut. I did not know whether I had a cyst

or an abscess, but always understood that a cyst was pale in color. I realized it was a dangerous place to incise, but I made an incision, and immediately a great deal of pus gushed forth. I do not know whether the patient coughed in my face or whether the pus was under unusual pressure, but it came with force on my face and neck. The patient was immediately relieved. Further examination showed that the swelling involved the aryepiglottic fold and the lower side of the larynx. It was about a week before all the tumefaction disappeared from the base of the tongue. Two weeks after incising this abscess, I was called by the patient, who said that his throat had filled up again, but that he was not suffering as much as he had formerly. I had him come into the hospital. On palpation, I found a pedunculated tumor, overhanging one-half inch in the median line. While cocainezing, the abscess ruptured but only a small amount of pus escaped. The mass, having been seized with a tenaculum, was removed by means of a snare loop. I realized that I was operating in a very dangerous place, for the abscess was situated in a position similar to that shown in Dr. Waugh's slide. There was not nearly so much pus as at the first operation. The sac was about the shape of the cap used on a test tube, the mouth being three-fourths inch in diameter. Before removal it was glistening, shiny and red; after removal it looked like lingual tonsil tissue. There was very little bleeding during the operation. The sac wall was one-fourth inch in thickness. The pathologic report was that the tissue consisted of lingual tonsil, without evidence of a cyst wall. The content was pus. The patient has had no further trouble and has made a good recovery.

DR HERMAN B. COHEN, Philadelphia, Pa.: I wish to thank Dr. Waugh particularly for the opportunity of listening to his paper, because, since 1917, when I wrote an article on lingual tonsil, and reported a case of lingual quinsy, I have seen little or nothing about this condition. The patient was in a hospital for several days, got no relief, asked for his clothes and went to another hospital. There he got no relief, having been given hot gargles and external applications. He asked for his clothes and left. He went to another doctor, who referred him to me, and I found pathology similar to Dr. Waugh's description. I made a deep incision in the most tender spot, and got pus and immediate relief. Since then, I have had a case in April, 1922, and another in May, 1923. The second case was not difficult to diagnose, because pus was oozing out from the lingual tonsil. After simple incision, enlarging the pocket, the patient got well. In the third case, the patient looked as if he were in the last stages of tuberculosis. He was unshaven and very much emaciated. There was a history of ordinary sore throat, no tonsillitis or other pathology of an abscess of the tongue. At the hospital I found the tender spot, and under local anesthesia incised it, and the patient got relief almost immediately. This makes a total of three cases that I have seen in six years.

DR. W. W. CARTER, New York City: I think the Academy should be congratulated on having Dr. Waugh present this very important subject in this attractive manner. It is important, because I believe a great many more patients die from this disease than is suspected.

I think many men have done as I have, and not reported cases. I have seen three cases, the last one last Friday. If Dr. Waugh is going through New York, I shall be glad to have him see this last case, for it is a true case of lingual quinsy. This man claimed to have been sick for two weeks of this disease. When he came in, the tongue was so swollen that it filled his whole mouth. Underneath the tongue was an extreme passive congestion, and there was a discharge of thin, flocculent pus, which seemed to be coming from the base of his tongue on the left side. His temperature was 103° F., he was suffering a great deal from dyspnea, and evidently had some edema of the epiglottis. By manipulation, I could see the uvula, which was very edematous. This differed from the case reported by Dr. Waugh. He said the uvula in his case was not edematous. I saw that something had to be done at once. I operated, and afterwards saw exactly the same appearance in that man's tongue that Dr. Waugh showed us on the screen. I was afraid to go in deeply with a knife, because the anatomic landmarks were obscured by the congestion and swelling, and there was great danger of wounding an important vessel or nerve, and I feared terrific hemorrhage. So we prepared to tie the common carotid if we got any bleeding. I took a long, fairly blunt pointed scissors, put them under the tongue on the left side, went right along the base of the tongue with them closed, and then opened them gradually. There was an immediate discharge of very foul pus, which was disposed of by the suction apparatus. Whether the man will get well or not I do not know. The other two patients died from edema of the glottis.

I think this subject is very important. I believe Dr. Mayer made the suggestion, and Dr. Waugh referred to the fact, that these cases should be operated from the outside, because we do not get the drainage from the lowest part from the inside. I shall be very glad, indeed, to have Dr. Waugh or any of the rest of you see this case in New York.

DR. H. P. MOSHER, Boston, Mass.: It seems to me that cases of abscess of the tongue are divided into two classes. Most of us, on thinking back over our clinical experience of a number of years, can recall cases of abscess of the base of the tongue which were relatively simple surgically; not simple from the patient's point of view, for the patient was very uncomfortable, but surgically they were. Such abscesses at the base of the tongue are readily evacuated by an incision under the tongue, and the evacuation is not accompanied as a rule by any dangerous complications. This, I feel, is well established. Whether the infection in these simple cases comes from the lingual tonsil I do not know, but as Dr. Waugh has looked up the literature, and thinks so, he is probably right.

In the second type of abscess at the base of the tongue, there is marked infiltration of the tissues of the neck, either in the midline or on the side. Such cases should be watched, knife in hand. At any moment there may be an infection and thrombosis of the internal jugular vein. If this happens, unless the internal jugular vein is dealt with from the side of the neck, just as thrombosis of the lateral sinus is dealt with, the case is very likely to end fatally from septi-

cemia. It has been customary to label these cases deep abscess of the neck from septicemia, and to neglect to find out the condition of the vein.

Recently, I saw a doctor's son who had a marked infiltration of the neck below the jaw in the midline. This followed an attack of acute tonsillitis a week before. The case seemed a typical Ludwig's angina. An external incision was made in the midline and carried to the base of the tongue, where it evacuated a very foul abscess. This extended up on the left to the region of the left lingual tonsil. The tongue did not get back to normal for a week, but the convalescence was normal.

External incision in the midline of the neck, I think, is an ideal way to deal with abscess at the base of the tongue, where there is infiltration of the neck in the midline. Where there is infiltration on the side of the neck, the lateral route is preferable. I do not share Dr. Waugh's fear of incising under the tongue in the first type of case.

I should like to ask Dr. Waugh whether or not the condition of the internal jugular vein was determined at the autopsy on his case.

DR. HARRY L. POLLOCK, Chicago, Illinois: I agree that there are two types of tongue abscess. Of the first type I have seen several. I do not think they are quite as rare as the essayist thinks the other type is. I have recently had the case of a confrere, who had an abscess of the base of the tongue that was of about ten days duration. He was living several miles from Chicago, and I was leaving the city and could not do anything. About a week later, he came in again. The abscess had burst spontaneously, but I could still see the pus coming out. These cases can be opened up easily and drained.

Of the Ludwig type, I recall, I think, but two cases. The trouble is you do not know just when to begin to open them. You do not wish to go in externally, for it is quite a task, and the infection follows the deep fascia right down. In one case, the patient got relief from the abscess symptoms, but died from a mediastinal abscess. The other patient also died. I think all these cases where they progress so far have died. I do not know whether it is the early treatment or what. I think, in the last few years, I have seen ten of the small abscesses. I do not know whether this is the type we get early and open early, or whether it is a different type of infection, but I have never seen any case recover when it has gone so deep and there was so much destruction as the case the Doctor showed on the screen.

DR. THOMAS E. CARMODY, Denver, Colorado: I, too, believe with Dr. Mosher that there are two distinct types. They are those that are somewhat chronic, as I would call them, with abscesses in the region of the tonsil. We also have many smaller abscesses in the base of the tongue. I think in the other type, which, as Dr. Mayer stated, are very much like the Ludwig's angina, the diagnosis is very difficult between these conditions until operation. I have had two cases which I have operated successfully, both from the outside. I had a case a number of years ago that descended into the mediastinum before I saw it, and it was not opened early enough, or I did not open it thoroughly enough down along the jugular, and that case

resulted fatally. In another case, I opened through just the same as we would a Ludwig's angina, with a large Kelly forceps, and was able to drain with tube. It was not necessary in that case to make the wide incision. I saw another case, in consultation, with much extension down along the palatopharyngeal to the larynx, which we were able to open from the inside. In another case such as Dr. Pollock mentioned, which I saw with Dr. Bane, the abscesses would come on every few months and rupture spontaneously. Whether the condition followed an infection of the tonsil or not I do not know, but they frequently do follow an infection of the lingual tonsil.

DR. J. M. WAUGH, Cleveland, Ohio, (closing): I think Dr. Mayer's suggestion that this may have been an extension downward from the previous tonsillitis is important. A considerable interval of time had elapsed since the previous tonsillar attack. Had a culture been made from the tonsils at the previous attack of quinsy, the streptococcus viridans might have been demonstrated, and the urgent need of operation impressed on the patient. This infection was probably carried in the tonsils during the interval between the acute attacks.

I do not think the lingual abscess was due to an embolism, but to direct extension from the lingual tonsil infection.

The case reported by Dr. Butler I do not think was related to lingual quinsy. It probably was an infected cyst in the pyriform sinus.

Lingual quinsy should not be confused with Ludwig's angina. They are two entirely different diseases, both in origin and character of extension, and Ludwig's angina cases call for prompt external drainage. Ludwig's angina follows along facial planes in the neck, whereas lingual quinsy is a deep seated localized process. The ideal drainage in the latter group should be from inside the mouth, if possible. An attempt to locate the abscess might be facilitated by an aspirating needle.

Most of the small abscesses seen in the tongue are due to broken down tuberculous nodules, or to degenerating syphilitic gummata. These are usually located anterior to the circumvallate papillae.

The case reported by Dr. Carter interests me very much. If there is no blood stream infection, his case will probably recover.

I am pleased to note the experiences of other members of this society with this rather rare form of quinsy, and hope before long to collect these into one report.

THROUGH-AND-THROUGH DRAINAGE IN THE TREATMENT OF FRONTAL SINUITIS.

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Dissatisfaction with the results of the radical operation on the frontal sinus has led many rhinologists to employ it with increasing infrequency, and some have even abandoned it altogether, relying wholly upon suction or other nonoperative means for the relief of pathologic conditions in the frontal sinus. The reason for this attitude has been voiced on both sides of the ocean, and is perhaps adequately summed up by Howarth, the British rhinologist, when says¹:

"We know that by means of various burrs and rasps we can enlarge the frontonasal duct so that it will admit the passage of a No. 15 bougie..... but is it possible to maintain this opening patent, even with the daily passage of bougies, long enough to secure a permanent cure of the sinus suppuration?.... The opening that one can make is not a very big one, and as it is achieved by rasping the bony wall of the duct, the raw surface of the bone naturally forms granulations which tend to become edematous and to obstruct the passage. In some cases new bone is formed from the cut surface, and may result in a stenosis which is far more troublesome to deal with than the original obstruction."

It is the belief of Thomas J. Harris², that most of the failures following the employment of the Killian operation—the aim of which is to obliterate the sinus—are due to reinfection, because the sinus is not completely obliterated. This is practically always owing to one of two causes; either the diseased contents of the sinus or the ethmoid are not completely removed, or the soft tissues of the orbit do not proliferate sufficiently to fill the sinus cavity, because of the removal of some part of the floor of the sinus. A direct consequence of this extensive destruction of the floor is the sinking inward of the outer wall of the nasofrontal canal, resulting in pronounced narrowing, even to atresia, of the passage.

The same difficulty has confronted the French rhinologists, for Mouret³ remarks that his chief objections to making a

large orbitonasal resection are the fact that the soft tissues sink toward the sinus so that they come in direct contact with the cavity (together with any possible focus of infection remaining after the surgical intervention), and the further likelihood of resulting stenosis of the frontonasal duct.

It was to overcome these difficulties that Lothrop⁴ devised the operation which he described in 1915. Making a small opening in the anterior wall of the sinus, the diseased tissue is curetted away, and rasps and burrs introduced to enlarge the nasofrontal canal as much as possible. Thus, drainage and free ventilation are secured, and provided the opening remains patent, the results should be uniformly good.

The three principal reasons for failure in operations on the frontal sinus are:

First: An inadequate nasal opening.

Second: Closure by cicatricial contractions of an apparently adequate intranasal opening.

Third: Failure to free the sinus of infected polypoid tissue and carious bone.

A comparatively small nasal opening is ample *if it remains patent*. Even if a good deal of polypoid tissue may be left within the sinus, it will disappear if the permanent nasal opening is sufficient to permit free ventilation and drainage.

Our main problem, therefore, is to prevent the cicatricial closure of the intranasal opening. The swelling of the tissues around a newly created opening incident to operative traumatism, in addition to the constant bathing in pus from the infected sinus above, makes a fertile soil for the production of exuberant granulations and, eventually, adhesive bands across the floor of the sinus. I believe, both from a theoretic standpoint and from practical observation at secondary operations, that periosteum may be floated out on these granulations both in front and behind, until union is completed and actual new bone formation takes place.

It was to prevent this outcome, that I began to use drainage tubes of various sorts. I first employed the Ingals gold flange tube after intranasal frontal operations, and later a similar intranasal tube, either an Ingals or rubber tube, after the external operation. I have now abandoned all these in favor of through-and-through tube drainage following the Lothrop operation, especially when there is a bilateral frontal involvement.

The Lothrop technic must be carried out carefully, with spe-

cial reference to the complete rasping away of the posterior projecting bone from the anterior wall.

The rasp or burr is directed as much toward the septum as possible, to prevent injury to the surrounding ethmoid cells if any of these remain healthy. After the operation is completed, a rubber catheter, usually a 20-French, is inserted into the external wound through the sinus and into the nasal cavity. About one inch of the tube is left projecting from the external wound, fixed with a safety pin. The section of the tube within the sinus is perforated in several places and extends into the nasal cavity, three or four inches, in the posterior direction. Before the patient leaves the operating room, both tube and sinus are thoroughly flushed with warm normal saline solution, in order to prevent obstruction of the tube from blood clots and tissue debris. The

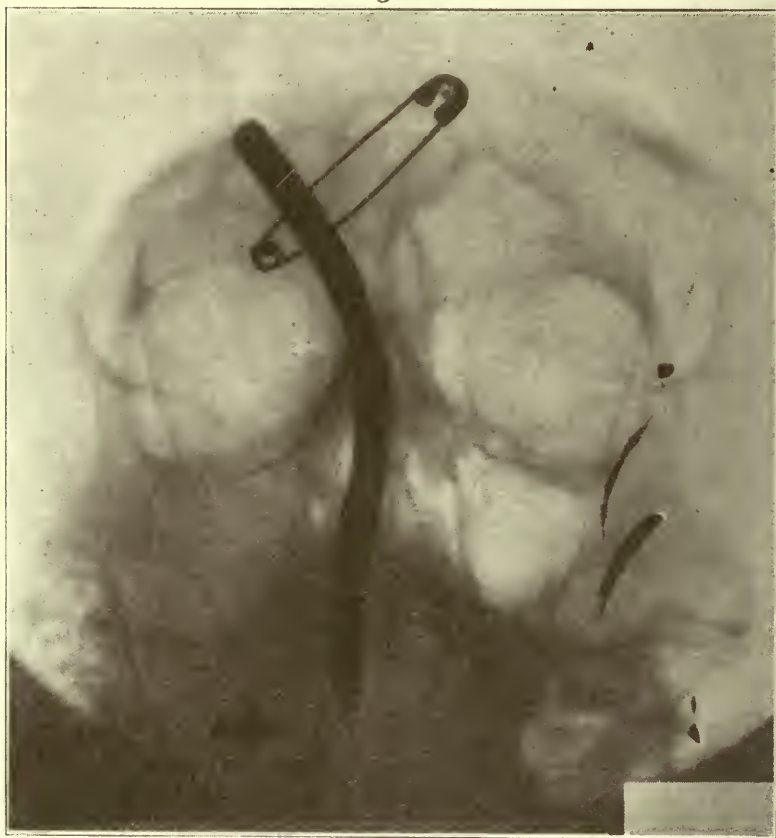


Fig. 1. A-P. exposure showing tube in position.

nurse is instructed to repeat this flushing every hour for the first twenty-four hours after operation. Medication may be put directly into the sinus after each flushing, the particular medication employed depending upon individual choice or the peculiar requirements of the case. I usually find some one of the organic silver salts to be satisfactory.

After the first day, the number of irrigations may be reduced to one every three or four hours during the day, and one at night. The patient quickly learns the method and can tell whether or not the tube is clear, by the "salty taste" in the mouth. He is permitted to leave the hospital at the end of the first week, being told to continue the irrigations three or four times a day. A soft rubber ear syringe may be effectively used, either by the patient himself or a member of his family. A gauze and adhesive dressing is applied over the external wound and tube, so that he may go about his business, reporting daily at the office or hospital.

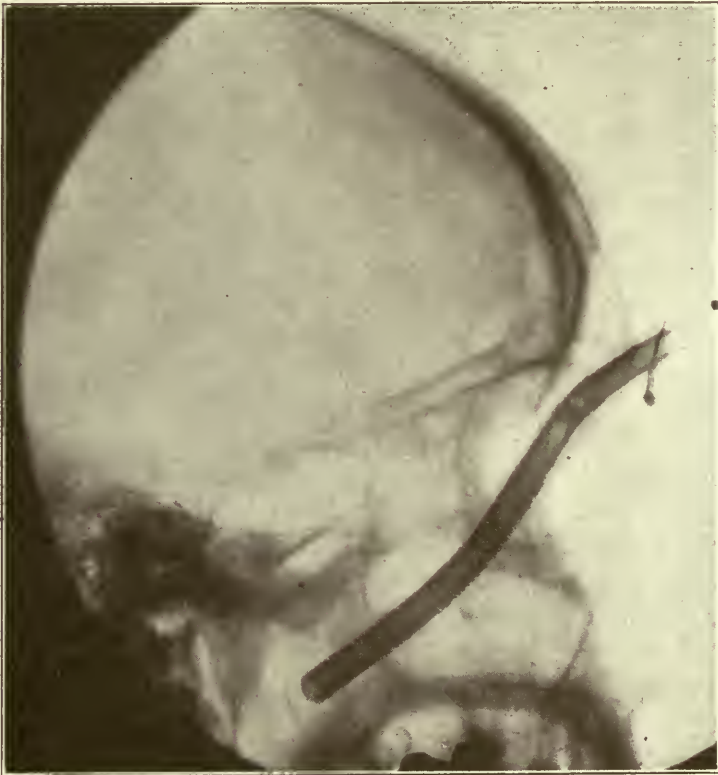


Fig. 2. Lateral exposure showing tube in position.

At the time of these daily visits, the tube which extends posteriorly in the nasal cavity, is grasped and brought forward through the nostril, and the sinus irrigated. These excursions of the tube prevent adhesions from forming in the anterior angle, and assure an opening even larger than the actual size of the tube.

By far the most important feature of through-and-through drainage is leaving the tube in position for a sufficient length of time. It should never be removed until at best three weeks have elapsed, and if the case is unusually chronic or severe, should be left in position for five or even six weeks. After the tube is taken out, the edges of the external wound are closed by adhesive plaster or by a single suture stitch. Should a small external fistula still remain, irrigation may be continued through this opening for a few days longer.

Skilern⁵ calls attention to the fact that the persistence of the discharge is proportionate to the extent of the surgery employed. Much regeneration of tissue must occur before the parts again assume their earlier functioning activities. The mucoid discharge "represents Nature's attempt to throw off those portions of tissue which have been devitalized by our curettes and cutting forceps."

The intranasal opening will now be a large firm ring of scar tissue fairly free from granulations, and this should be consistently left alone. Any attempt at dilatation or instrumentation of any kind is absolutely contraindicated. In fairly large noses, the sinus can be inspected directly by inserting a small laryngeal mirror into the nasal cavity. The external scarring resulting from this procedure, even when the tube is left in position the maximum length of time, is very slight, as is shown by some "close up" photographs.

Though I have employed some sort of tube drainage for ten years, my experience with this particular technic has extended over a period of only five years. I have operated in twenty cases, sixteen of which I have been able to follow up quite closely. The first patient was operated on in June, 1918, and the last in May, 1923. Fourteen operations were done for the relief of chronic suppuration; two for acute suppuration; three for vacuum sinusitis, and one for subacute suppuration. One chronic case was complicated by nasal trauma, another by a dermoid cyst, and still another by an extradural abscess.



Fig. 3. Maximum amount of external scar. This case was complicated by extradural abscess. Operated, Oct., 1921.



Fig. 4. Unusually small scar. Tube was left in position four weeks. Operated, May, 1923.

Summarising my experience in the accompanying condensed chart, my conclusions are:

First: Through-and-through drainage may be used after any external operation on the frontal sinus, except the osteoplastic or obliterating operations.

Second: Through-and-through drainage allows a firm dense ring of scar tissue somewhat larger than a 20-French catheter to form in the floor of the sinus, and prevents adhesive bands from bridging the opening and thus defeating the purpose of the operation.

Third: The tube should be flushed every hour during the first twenty-four hours after operation, i.e., the oozing stage.

Fourth: The tube must be left in position for at least three weeks.

Fifth: Medication can be put directly into the sinus.

Sixth: The employment of through-and through drainage is an extra precaution, insuring a greater percentage of good results after external operations on the frontal sinus.



Fig. 5. This shows about average scar to be expected. Operated, June, 1918.

Patient	Diagnosis	Duration	Previous Operations	Other Sinus Involvements	Result
Miss P.	Chro. Sup. L.	2 years	Septum, ethmoid L.	Ethmoid, L.	Good
Miss J.	Chro. Sup. L.	2½ years	Ethmoid, Internal frontal	None	Good
Sister C.	Chro. Sup. Bilateral	10 years	13 on all sinuses	All	No improvement.
Mrs. P.	Subacute Sup. R.	3 mos.	R. Middle Turb. inectomy	None	Good
Mr. S.	Chro. Sup. (Dermoid Cyst)	5 yrs. or more	None	None	Good
Mr. D.	Acute Sup. L.	2 weeks	None	None	Good
Mrs. F.	Chro. Sup. Bilateral	7 years	Bilateral radical ethmoidectomy	R. Antrum	Pain relieved. Some discharge even after antrum operation.
Mrs. P.	Vacuum R.	2 years	R. Ethmoid	R. Ethmoid	Good
Miss Mc.	Vacuum R.	2 years	R. Ethmoid	Ethmoid	Good
Mrs. K.	Chro. Sup.-L. (Extradural abscess)	3 years	L. Antrum	L. Antrum	No discharge. Some pain periodically.
Mrs. B. (Insane)	Chro. Sup. R.	4 years	Exter. drainage R. eye removed	None	Doubtful (State case)
Miss A.	Chro. Sup. L.	18 mos.	L. Ethmoid, Sept.	L. Antrum L. Sphenoid	Some discharge L. no pain
Mr. C.	Acute. R.	3 weeks	None	L. Frontal	Good
Mr. M.	Bilateral Sublatent (Arthritis)	5 years	Sept. R. Middle Turbinate	R. Antrum Sup.	Good
Mr. H.	Vacuum, bilateral	2 years	Bilateral. L. Ethmoid Intranasal L. Frontal	Ethmoids	Good
Mrs. K.	L. Chro. Sup.	4 years	Septum L. Middle Turbinate	L. Antrum Sup.	Good
Sister M. L.	Chro. Sup. L.	6 years	Septum L. Middle Turbinate	L. Antrum	Good
Lieut. P.	Chro. Sup. R. (Trauma)	1 year	R. Middle Turb.	None	Good
Mr. M.	Chro. Sup. R.	8 years	Sept., Bilateral Int. Frontal. Part Ethmoid.	Ethmoid L. Antrum R.	Pain relieved. Some discharging at intervals after expos.
Mr. D.	Chro. Sup. L.-R.	16 mos.	Anterior tip both middle turbinates.	None	Good

REFERENCES.

1. Howarth, Walter G. Operations on the Frontal Sinus. J. Laryngol. and Rhinol., Edinburgh, 1921, v. 36, p. 417.
2. Harris, T. J. Causes of Failure in the Radical Operation on the Frontal Sinus. J. A. M. A., Oct. 8, 1921, v. 77, p. 1147.
3. Mouret, J. Sur la Trepanation du Sinus Frontal. Rev. de Laryngol., etc., Oct. 15, 1920, v. 61, p. 561.
4. Lothrop, H. A. Frontal Sinus Suppuration with Results of a New Operative Procedure. J. A. M. A., July 10, 1915, v. 65, p. 153.
5. Skillern, R. H. The End Results of Radical Operations on the Accessory Sinuses. Ann. Otol. Rhinol. and Laryngol., March, 1923, v. 32, p. 139.

A MODIFIED RADICAL KILLIAN UNDER LOCAL ANESTHESIA.

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There are well recognized objections to and failures in the original radical frontal sinus operation, notably deformity and continued annoyance from reinfection. In the interest of better results, we offer "A Modified Radical Killian Under Local Anesthesia", the points of emphasis being—(1) Local anesthesia is perfectly satisfactory. (2) The technic is simple. (3) Very slight deformity. (4) Results satisfactory.

Several years ago, we observed an external frontal which had been done by removing the floor of the sinus, without consequent deformity. It occurred to us, that this was the cue we had long been looking for, so we extended this frontal sinus operation into a modified Killian. The bleeding in a radical Killian under ether has always been a great objection to us, as it necessarily prolonged the operation and made the procedure more or less uncertain. The only details of the technic we would stress in this operation is to remove completely the floor of the frontal sinus, the anterior, external and posterior walls of the nasofrontal duct, lacrimal bone, ethmoidorbital wall, and as much of the posterior part of the nasal process of the maxillary bone as necessary to make a *wide straight opening into the nose*, and in cases where the mucosa is greatly degenerated, to clean out thoroughly every vestige of the diseased tissue.

Anesthesia. If the patient is nervous or apprehensive, use a hypodermic of morphin, atropin and hyocin one hour before the operation. Prepare the nasal mucosa with cocain-adrenalin mud, and inject the supraorbital region with 2% novocain with a few drops of adrenalin added. If the antrum is to be opened, inject the infraorbital canal or the region of exit of the nerve with a similar solution.

Technic. Make a few short skin scratches at right angles to the brow to be used as a guide in closing the wound. Begin the skin incision a little past the middle of the brow (it can be extended later if necessary), and carry it down to the bone through the periosteum throughout the length of the incision. Begin the separation of the periosteum over the infundibulum of the frontal sinus, extending it under the floor of the sinus, over the lacrimal, nasal, maxilla, ethmoorbital wall as far as necessary. The bone incision is also begun at the infundibulum of the sinus. The ethmoids and sphenoids are attacked as in the regular radical operations, or the sphenoid wall may be dispatched through the nares. In some cases, the middle turbinate can be saved by pushing it against the septum. Now return to the intranasal part of the nasal process of the superior maxillary bone, and chisel away enough to remove the angle it presents to the inferior approach to the frontal sinus. This is an important item.

The interior of the nose may be dressed according to the operator's preference; we prefer vaselin or bismuth gauze lightly packed into the whole cavity for 18-24 hours. The skin incision is carefully and completely closed. Use a firm pressure bandage over the eye and brow for forty eight hours; then remove all external dressings. The intranasal wound is interfered with as little as possible. At the end of a week, pass as large as possible steel sound or olive into the frontal sinus, and repeat every 4 or 5 days until granulations cease to form. The pressure of the sound is more efficacious than curetting away granulations which may form.

Deformity. Ptosis of the lid gradually passes, and no external deformity results if the wound has been carefully coapted. In one case, there is 6° of hyperphoria after one year.

Results. We have cases of three years standing which continue free from suppuration. We do not offer this as a cure for all cases of pansinusitis, but we affirm that it has led to a happy solution of many cases in our hands.

DISCUSSION ON PAPERS OF DR. ROBERTS AND DR. CARPENTER

DR. HENRY B. LEMERE, Omaha, Nebr.: I think we are indebted to Dr. Roberts for the procedure which he has to-day advocated and explained to us, and which he uses in carrying out the Lothrop operation. I think it will make many frontal sinus operations a success which otherwise, without this through and through drainage, might be failures. I have had the great pleasure, previous to this paper, of seeing Dr. Roberts' cases in Kansas City and of hearing him tell of his procedure, and I wish to say, that from the cosmetic standpoint and also the curative standpoint, this procedure has been very satisfactory. I feel that through and through drainage in these sinuses is a tremendous advantage. I have advocated it in treatment after the antrum operation, and I think it is just as important in frontal sinus cases. It did not occur to me that it could be carried out after the frontal sinus operation, until I heard Dr. Roberts report his cases two years ago. I feel that the through and through drainage is important, in that it carries away the secretion, makes granulation tissue less likely to form, and, also, in Dr. Roberts' procedure, the movement of the catheter has the effect of lessening the granulation by direct manipulation. I think that the objection that we sometimes hear, that irrigation and through and through drainage produce a waterlogged condition of the sinus, is not warranted, and that the secretion, if left on the mucous membrane, will be much more likely to produce a waterlogged condition than will the water used in cleansing these cases.

I wish to mention that Dr. Carpenter's plate, in which he showed the double frontal sinuitis, has a mottled appearance which is very suggestive of an osteomyelitis, and I think Dr. Carpenter is to be congratulated in his result, for these cases sometimes go on to progressive osteomyelitis with a fatal outcome.

DR. J. J. KING, New York City: I wish to congratulate Dr. Carpenter upon the splendid results he has obtained in a large array of frontal sinus operations. The results as shown by his pictures are perfectly beautiful, and I think it is unusual for one man to be able to show so many successful frontal sinus operations. The operation seems quite rational. I think the large opening down in the floor of the nose is rational, and when the operation is carried out under local anesthesia, I think it increases the safety of the procedure, and renders convalescence quicker and the morbidity less.

First of all, I wish to state that I am very conservative about operating on frontal sinuses by this method. I resort to it only where the indications are very clear. I prefer to treat them in a conservative way if possible, especially by drainage and ventilation of the sinuses, and it is my experience, that the majority of these patients have recovered without my resorting to external operations. I have found it necessary to do only a small number of external operations, for by treating them conservatively most of my patients have recovered. We sometimes have to resort to these drastic operations when other measures, less heroic, have failed. I am thoroughly convinced that, in the long run, the majority of my patients are better served by conservative methods.

As to anesthesia, I am a strong advocate of local anesthesia for as many operations as can be carried out under it in our branch. I would add to the method described by Dr. Carpenter the preliminary administration of an anodyne in all of his cases. This can be carried out very readily in all frontal sinus operations where the cooperation of the patient is not desired. In some instances it is necessary to have the cooperation of the patient, but in working on the mastoid and the frontal sinuses, this cooperation is not essential. I have recently found that the combination of morphin with magnesium sulphat has been the best thing we have ever used, and I have had some experience with most of the drugs which have been recommended for this purpose—scopolamin, morphin, procain and all of the drugs. The results I have had from magnesium sulphat have been very satisfactory. I use one-eighth grain morphin dissolved in 2 mls of 25 per cent chemically pure sterile magnesium sulphat solution. I have used this for two years, and am convinced that this synergistic anesthesia is the best I have found.

Such a solution of magnesium sulphat has been prepared for me by the Metz Laboratories.

DR. HENRY B. HITZ, Milwaukee, Wis.: I was much interested in the paper of Dr. Roberts, because I have had some experience of a similar character in the past year. After having done numerous operations of various types and character upon the frontal sinuses, many of which have been only partial successes, and some of which I have had to do over again, I have come to the conclusion that through and through drainage is absolutely the right procedure. I have treated the matter in a somewhat different manner than Dr. Roberts. In a bad, suppurating, extensively developed sinus, one in which the disease had existed for quite a number of years, with bone softening and filled with granulations and pyogenic membrane, it was very difficult to make a clearing out of that sinus through a small opening, as I have had reason to observe in a number of cases during the past year.

In the first case that I shall mention, the patient had been operated twice in the army and once subsequently, with poor results. A suppurating sinus emptied from the angle of his left eye. I started with the intention of making a Killian, but upon exposing the floor of the sinus, the whole of the roof of the orbit was exposed clear back, and the outer table above the supraciliary ridge was completely softened. I concluded, that in a man who had very deep seated eyes and was very thin, I would get far better results with something other than a Killian. Consequently, I removed the complete outer table above the supraciliary ridge. I then used a Dakin tube through a counter opening in the forehead, carrying it back. After clearing out all the pyogenic membrane and bony trabeculae, and cleaning the sinus thoroughly, I placed a rubber tube as large as my finger in the ostium and went through the nose, and kept it in for six weeks. Through-and-through washing during that time was kept up daily, with the gratifying result of a complete cure. The operation left a dimple in the center of the forehead, but removed the other scars, and the man had a perfectly satisfactory recovery. Three others treated similarly gave the same satisfying result with a minimum of deformity.

These severe conditions in my judgment can safely be met only in

a truly surgical manner, and I believe the method presented by Dr. Roberts fulfills this ideal.

DR. WILLIAM MITHOEFER, Cincinnati, Ohio: It is interesting to note, in talking with various colleagues, that so few men do the radical frontal sinus operation. I, personally, was prejudiced against this operation in my early days of practice, and always approached the subject from a preconceived point of view. I was like the man who was unable to see the forest on account of trees. In the early days of practice, I rarely did the radical operation, but finally decided that something had to be done for patients with severe headache, where intranasal measure had failed. I started with the Killian, but was not satisfied, so went on to the Lothrop, and eight out of twelve had complete closure of the nasofrontal duct after operation. I then used the orbital route, as recommended by Ritter, and this has given me the best results. I have done over forty of these operations under local anesthesia, and in the last few years have had much better results than formerly. The nasofrontal duct is bound to close unless you make a mucous membrane flap from the lateral wall of the nose. The flap is made as the first step of the operation. In order to make the flap, the knife is inserted high up on the lateral wall, and the mucous membrane incised along the entire length of the wall as far anterior as the apertura pyriformis. From the anterior end of the incision, another one is made along the lateral wall to the anterior part of the inferior turbinate. Another incision is made joining the upper end of the first incision, and extending along the lateral wall to the region opposite the middle turbinate. The mucous membrane is dissected downward, and allowed to remain in this position during the operation. As a final step of the operation, it is gently packed into the orbit. A drainage tube is also inserted into the nasofrontal duct, and allowed to remain fourteen days if possible. This method of operating has given us the best results.

DR. JOSEPH C. BECK, Chicago, Ill.: All the gentlemen speak of getting results, and do not consider that it is still necessary to do the radical operation, and that the failure is due to an existing osteitis. I think we can test out an operation only by means of the X-ray examination.

The thing I wish particularly to speak of is the synergistic anesthesia of Gwathmey. We have used this over one thousand times. In addition to the morphin and magnesium sulphat, we use ether in small amount by rectum, with some such medicine as paraldehyd. This is very important in this connection. I am surprised that some of the New York men did not mention this anesthesia, for it was first called to our attention by Gwathmey of that city.

I believe Dr. Roberts' idea of obtaining the permanent opening of the canal is excellent, and should be attempted by everyone.

DR. KATE W. BALDWIN, Philadelphia, Pa.: I simply wish to state that Dr. Walter Freeman of Philadelphia taught us through and through drainage some years ago. As he is gone, it seems only just that his work should receive the recognition it deserves.

DR. THOMAS E. CARMODY, Denver, Colo.: Speaking of priority, an operation similar to the so-called Lynch operation was described by Gallagher, of Denver, in 1910, and published by him in the *Laryngoscope*, April, 1911.

DR. SAM ROBERTS, Kansas City, Mo. (closing): I do not know of the article Dr. Baldwin mentions, although I made a good search of the literature.

Regarding Dr. Lemere's suggestion that the tissues might become waterlogged, I will say that I use only a small amount of normal saline, just enough to keep the tube clean.

I agree with Dr. King's remark regarding Dr. Carpenter's paper, that radical sinus operations should be rarely done. You perhaps noticed in my charts, that most of the patients had had one or more simple procedures first. I do not consider the Lothrop operation particularly radical; it is the most conservative external operation. By using the through and through tube, I have overcome the greatest cause for failure.

DR. E. W. CARPENTER, Greenville, S. C. (closing): Many details of the operation were omitted in the paper for the sake of brevity. We never employ this radical external type of operation if satisfactory results can be produced by an intranasal operation.

Simple mastoids can be performed under local anesthesia with as much satisfaction and success as the radical intranasal sinus operations are done.

SUPPURATIVE DISEASES OF THE LUNG; BRONCHOSCOPIC DRAINAGE AS AN AID TO TREATMENT BY THE INTERNIST.

CHEVALIER JACKSON, M.D., Sc.D., F.A.C.S.

PHILADELPHIA, PA.

The purpose of this paper is to present a few broad generalizations based upon the bronchoscopies done for various suppurative diseases of the lung due to conditions other than foreign body. The bronchoscopies were done by my associates, Drs. Gabriel Tucker, Louis H. Clerf, Robert M. Lukens and William F. Moore, at the three divisions of the Bronchoscopic Clinic, namely, at the Jefferson Hospital, at the University Hospital and at the Department for Diseases of the Chest, Jefferson Hospital. The patients were selected and treated in consultation with the groups of internists and surgeons associated with the Jefferson and University Hospitals respectively, and acknowledgment for hearty cooperation is due to Drs. Thomas McCrae, Alfred Stengel, David Riesman, Elmer H. Funk, Edward E. Graham, J. P. Crozer Griffith, E. Quinn Thornton, Henry K. Mohler, G. H. Perry Pepper, E. J. G. Beardsley, J. Chalmers Da Costa, John H. Gibbon, George P. Muller, D. L. Despard, F. J. Kalteyer, R. A. Kern, J. Blechschmidt, Ralph L. Engel, T. Grier Miller, Joseph Sailer, T. G. Schnabel, and others.

Detailed reports of cases will be omitted for two reasons: 1. Reports of concrete cases would detract from the brevity and clearness aimed at here; 2. Detailed reports of individual cases have been¹ or will be² presented by each of my above mentioned coworkers. A steady stream of letters coming to us at the Bronchoscopic Clinic demonstrates the urgent need for a general focussing of the subject, based upon experience up to the present time, to serve as a guide until modified by the further accumulation of data. Ultimate conclusions in medicine are never possible because of never ceasing progress. Our accumulation of data, however, has enabled us to present very definite conclusions.

Nontuberculous and nonmalignant suppurative diseases of the lung may be broadly classified, from an anatomicopathologic viewpoint, as purulent bronchitis, drowned lung, abscess, and bron-

chiectasis; but it must be remembered that clinically these conditions so often coexist that differentiation is impossible. Moreover, there is much overlapping with other conditions such as asthma, benign growths, compressive stenoses, etc. It must be stated, therefore, that separate consideration here is only for convenience of literary consideration. Clinically these cases are grouped as "lung suppuration" and are treated according to lesions found at the diagnostic and subsequent bronchoscopies, after consultation with the internist, the roentgenologist and the surgeon.

Lung mapping as first advocated by the author³ is very useful medically and surgically. It is done by means of a stereoroentgenogram taken promptly after the bronchoscopic insufflation of dry powdered bismuth subcarbonat. The walls of the bronchi are thus beautifully mapped out in the stereoroentgenogram. This

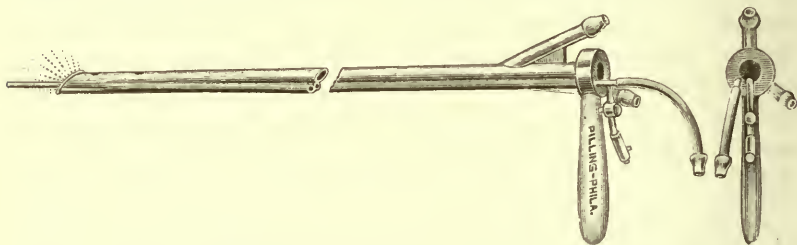


Fig. 1. Special irrigating bronchoscope, recently devised by Dr. Robert M. Lukens, of the Bronchoscopic Clinic, for irrigating pulmonary suppurative cavities drained by bronchi too small to admit the tip of the bronchoscope. The irrigating tube is passed down through a special channel, and can be extended three cm. beyond the beak. The lumen of the bronchoscope is not encroached upon by the irrigator, leaving a clear view of the draining bronchus as it is being irrigated.

is of great usefulness in the localization of foreign bodies and the mapping out of bronchiectatic areas. The bronchi stand out like the branches of a snow covered tree. The bismuth in all cases has entirely disappeared in two or three days, and has proven quite harmless at the Bronchoscopic Clinic. In nontuberculous pulmonary abscess viewed endoscopically, as shown by the author⁵, there is rarely a cavity as seen in advanced tuberculosis. Usually, the abscessed area is filled more or less completely with granulations. In this condition, for localization some may prefer the method used by my distinguished pupil and co-worker, the late Henry L. Lynah⁴, working in conjunction with the eminent roentgenologist, William H. Stewart. About 8 c.c. of a suspension of bismuth subcarbonat in olive oil, sterilized by boiling, is slowly injected into the abscess. "When it enters

lobular lung structure, it is noticed as a dull, opaque area; when it enters the abscess cavity, the fact is recognized by the metallic luster."

Among the advantages of the dry powder insufflation seem to be the perfect way in which the normal bronchi are differentiated (Fig. 10), the prompt expulsion of the powder, and the freedom from any tendency to form compact masses.

Indications. Bronchoscopy is indicated in any case of suppuration of the lung, for diagnosis. In cases not due to foreign body, of which there is rarely a negative certainty, delay for general study and a test of the efficiency of general medical treatment is often advisable; but in cases of foreign body known or suspected, bronchoscopy is the primary and most important indication.

Contraindications. Recent hemorrhage is a contraindication, dependent upon the amount of blood expectorated. Blood streaked sputum may mean only oozing from granulations; if so, it is no contraindication. The expectoration of clots, or of blood in quantities of a few cubic centimeters, may mean the beginning erosion of a vessel, in which case bronchoscopy is contraindicated until a week or two after the blood has disappeared from the sputum; not because of any risk of tearing open a leaking vessel, but because of a certain degree of coughing, straining and increased blood pressure during bronchoscopy, which would render delay for healing of the eroded vessel advisable. On the other hand, bronchoscopy may be indicated in certain cases for the arrest of severe pulmonary hemorrhage.

PULMONARY ABSCESS.

In the diagnosis and treatment of pulmonary abscess, the bronchoscope has proven itself of the utmost importance. In many cases, the previous erroneous diagnosis has been corrected by it, and many cases also have been cured by the local drainage and local medication afforded by its use.

Diagnosis. The diagnosis of the presence of a fully developed pulmonary abscess is readily made by the roentgen ray and the physical signs. The differentiation between abscess, bronchiectasis and neoplasms is not always so easy, and in many cases is impossible without the aid of the bronchoscope. Bronchoscopically, the differentiation is, in most cases, readily made by the dilated state of the bronchi, which are often ulcerated, in bronchiectasis. In malignancy, the endoscopic appearances and a bronchoscopic-



Fig. 2. Roentgenogram of chest of woman, aged 27 years. Pulmonary abscess of right upper lobe. Severe cough with profuse foul expectoration began 8 days after cholecystotomy under ether anesthesia. Intermittent productive coughing paroxysms. Bronchoscopic treatment began 6 months after onset of suppuration. In the lower portion of the upper lobe, there is seen a large area of exudate which is not adequately drained. Arrows surround the cavity.



Fig. 3. Roentgenogram of same patient as shown in Fig. 2, made after fifth weekly bronchoscopic treatment. This plate shows only a very small amount of exudate in the parenchymal portion of the right upper lobe, along the interlobar pleura. The abscess cavity is well drained. At last bronchoscopy, after 17 bronchoscopies, the bronchi were found to be free from pus. Physical examination of chest revealed nothing abnormal. Patient treated by Drs. R. M. Lukens and W. F. Moore at the Bronchoscopic Clinic, Department for Diseases of the Chest, Jefferson Hospital.

ally removed specimen of tissue will decide. The differential diagnosis between *drowned lung* and lung abscess often cannot be made with certainty, because without bronchoscopy it is not always possible to recognize the exact time when the breaking down of the normal walls begins. This breaking down is easily recognized by the bronchoscope, except in case of a very small abscess at the periphery of the lung. In these cases lung mapping as previously mentioned is essential.

Prognosis. In the bronchoscopic treatment of pulmonary abscess of other than foreign body origin, it must be remembered that we are dealing with a very chronic disease, which by any method of treatment, climatic, postural, therapeutic or surgical, requires in most cases a long time to obtain results. When a surgeon does a thoracotomy for pulmonary abscess, he does not expect, and never obtains the prompt cicatrization that follows the evacuation of abscesses in many regions of the body. The ramifications of the suppurative processes and the structure of lung tissue prevent such a result. It is well to remind the patient and the practitioner who has referred the patient of these facts, in order that they shall have no unreasonable expectations.

In some cases the results have been positively brilliant; in a few cases a few aspirations were all that was necessary. In two cases one aspiration turned the tide toward recovery.

There is an enormous difference prognostically between pulmonary abscess of foreign body origin and that of other origin, for instance the postpneumonic abscess. The explanation of this probably rests upon a different sequence of pathologic processes. In foreign body cases at the Bronchoscopic Clinic, the abscess has entirely healed and the patient has regained perfect health in 98 per cent of the cases, even after many years sojourn of the foreign body (in a number of cases over 20 years, and in one case 36 year). As explained in the Mütter Lecture⁹, the removal of the foreign body and its accompanying granulations permanently improves drainage. Another factor is, that the onset and development of the suppurative process is slow and is resisted by the surrounding actively healthy tissues; whereas in postpneumonic abscess, the process is surrounded by an extensive area of tissues of low resistance, because they have been devitalized by the pneumonic process. In posttonsillectomic abscess, we have either a septic embolus⁵ or an aspirated virulent infective process⁶.

Therefore, it may be said that, while in foreign body abscess

the prognosis is almost uniformly good after removal of the foreign body, in postpneumonic, postinfluenzal and posttonsillectomic abscess we have a serious disease of only fair prognosis. If there are multiple foci, more or less isolated from each other, the prognosis is less favorable by any method of treatment. If the patient's resistance is high and he responds to treatment, the prognosis is good; if, on the other hand, the patient steadily loses in weight and strength, with a progressive increase in septic symptoms, the prognosis is bad. As to the local factor in prognosis, it may be said that a single cavity readily accessible from a large bronchus is curable in almost all cases, whereas in multilocular cases, with many small devious fistulous passages, blocked with granulations, leading to some but not all of the foci, the prognosis is not so good; but even in these cases, careful search with the "velvet-eyed" aspirating tube will usually discover and drain every focus and result in cure, if the patient has any considerable degree of recuperative power.

The best gauge by which to determine progress on which prognosis may be based is a chart such as we use at the Bronchoscopic Clinic, devised by Louis H. Clerf. It shows graphically the body weight, the leucocytosis, the temperature and the amount of expectoration. In Fig. 11, we have a favorable trend. Had a downward trend rendered it evident that we were not getting improvement from our treatment, it would have been discontinued.

As to the danger of bronchoscopic treatment, it need only be said that of the hundreds of treatments at the Bronchoscopic Clinic, no patient has died of any cause whatever within 2 weeks of a bronchoscopic treatment, so that it may be said there is nothing to fear in the way of mortality from the bronchoscopic treatment of pulmonary abscess. It is, of course, to be expected that some day a patient in whom hemorrhage is imminent will be bronchoscoped on the eve of a hemorrhage, but in such an event it would be unwise to look upon the hemorrhage as a result of bronchoscopy. If hemorrhage were likely to be caused by bronchoscopic treatment, surely an instance of hemorrhage would have occurred in some one or more of the many cases treated for periods varying from 2 months to 2 years. In all, over 4500 treatments were given, without hemorrhage or any other serious complication.

The prognosis of *drowned lung* is always good if the bronchial obstruction can be relieved. In autodrownage from cadaveric

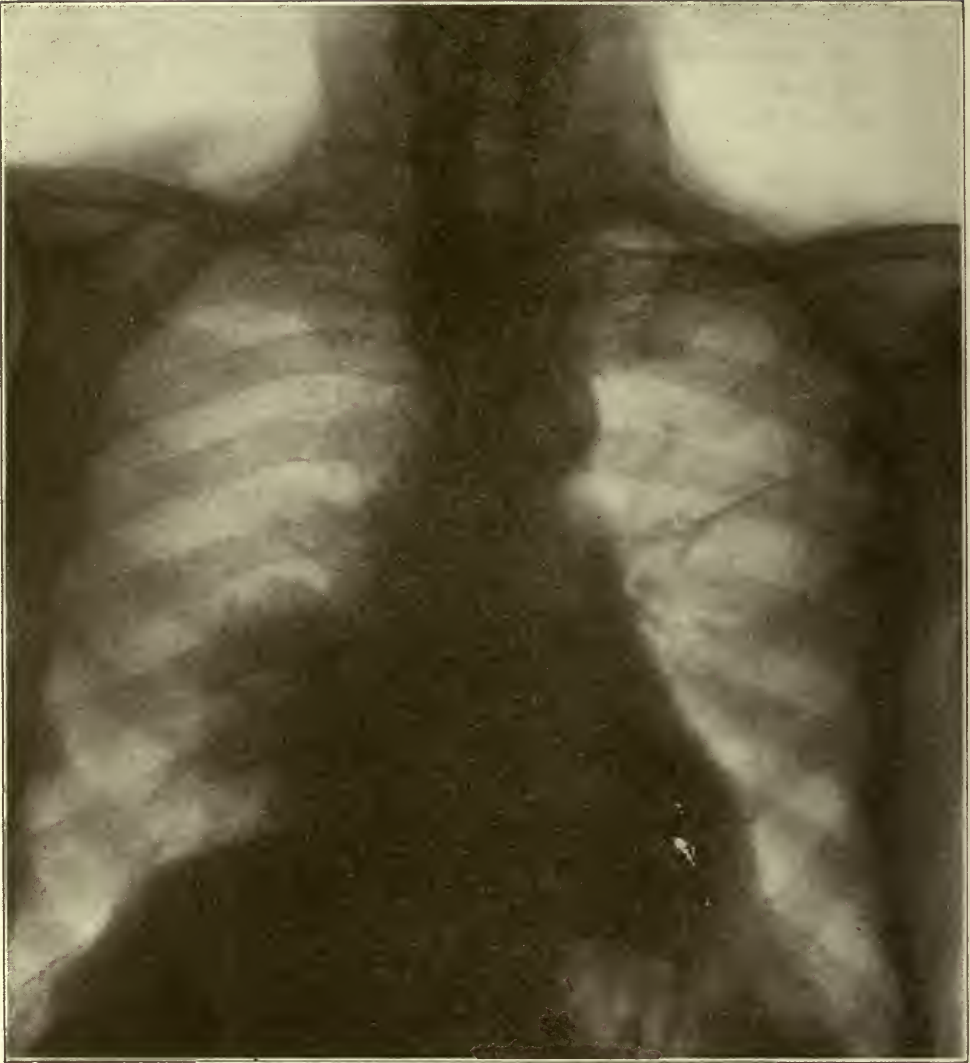


Fig. 4. Abscess of the middle lobe secondary to lobar pneumonia in a man aged 50 years. Diagnostic bronchoscopy revealed obstruction of the orifice of the middle lobe by granulation tissue. Following four additional bronchoscopies done at weekly intervals, the patient was discharged symptomatically well. Patient was treated by Dr. Louis H. Clerf, Bronchoscopic Clinic, Jefferson Hospital.

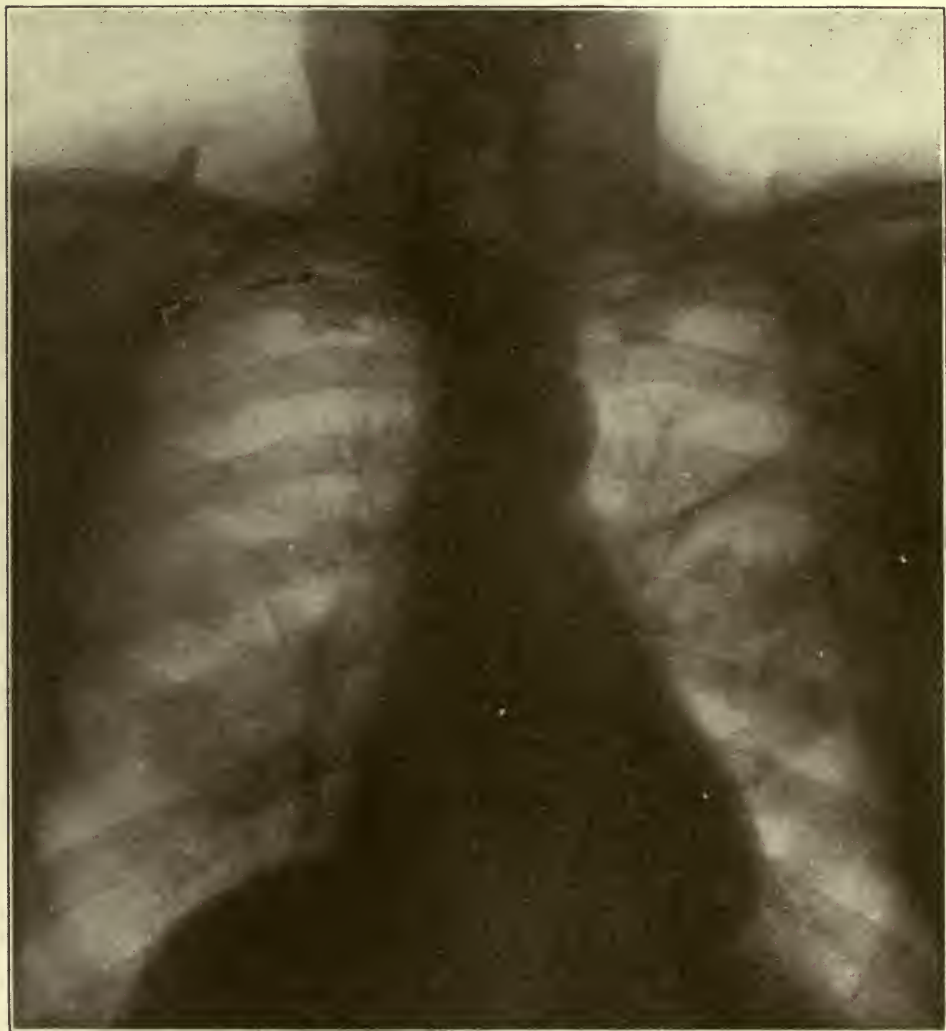


Fig. 5. Roentgenogram of patient with abscess of middle lobe, shown in Fig. 4. Roentgen ray examination made four months after patient was discharged from hospital. No Roentgen ray evidence of disease. There has been no recurrence of symptoms. Patient has gained 43 pounds in weight.

recurrent laryngeal paralysis, the prognosis is good if the lesion causing the paralysis is not of itself fatal.

Technic of Bronchoscopic Treatment. The first step in most cases is the improvement of drainage. Obstructive granulations should be removed with crushing, not biting, forceps. The regular foreign body forceps are best for this purpose. Caution should be used as to removal of the granulations with which the abscess "cavity" is filled in many of the chronic cases. After the removal of granulations, we usually commence weekly treatments, clearing out of the purulent secretions by aspiration with the aspirating bronchoscope and the independent aspirating tube, the latter being inserted into passages too small to enter with the bronchoscope. This is followed by medication applied by either swabbing or injection. Both methods have yielded good results. For swabbing we have used with excellent results silver nitrat, 10 to 20 per cent solution, as advised by E. Quinn Thornton. Good results have also followed applications of argyrol, 20 per cent, zinc sulphat, one-fifth of one per cent, and gomenol, 20 or 30 per cent in oil. For injection, from 5 c.c. to 20 c.c. of the following medicaments have been used with good results: argyrol, 1 per cent watery solution; silvol, 1 per cent watery solution; iodoform, 10 per cent oil emulsion; guaiacol, 10 per cent solution in mineral oil; gomenol, 20 per cent solution in mineral oil; bismuth subcarbonat suspension in mineral oil. Robert M. Lukens¹ and William F. Moore² of the Bronchoscopic Clinic report excellent results in posttonsillectomy abscesses from one tenth of one per cent phenol in normal salt solution, with the addition of 2 per cent Lugol's solution. Chlorinated solutions are irritating, and if used, require copious dilution. Liquid petrolatum, is, we think, to be preferred as a vehicle. Our experience at the Bronchoscopic Clinic with hundreds of cases shows conclusively that vegetable substances set up a violent reaction in the lung, whereas mineral substances do not. Nut kernels contain a large amount of oil. and while we have not yet been able to demonstrate positively that it is the oil that is the irritating principle, we have thought it best to use mineral oils in the treatment of lung suppuration not due to foreign body. Experience has shown that mineral oils thus used have been nonirritating. It must be remembered, however, that only a small quantity is used, never more than will cover the diseased area. To drown the healthy air cells with a deluge of oil would be uncalled for and probably injurious. In some instances, a little oil of eucalyptus has been added to the oil. The technic is quite simple. The aspirating

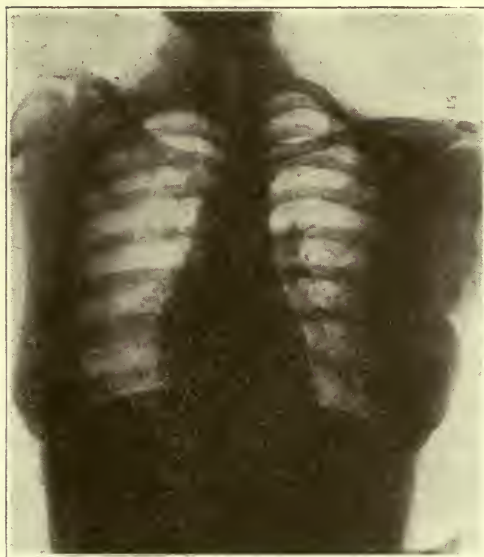


Fig. 6. Mrs. M., aged 27 years. Roentgenogram made November 27th, 1922, shows pulmonary abscess in right lower lobe, which developed after tonsillectomy.

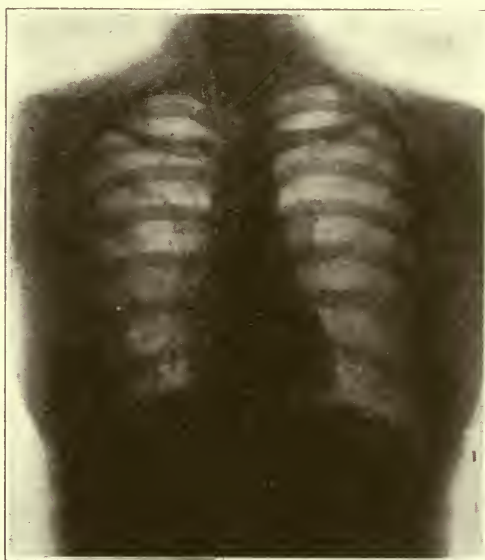


Fig. 7. Shows same patient as in Fig. 6. Roentgenogram made 8 months later. Only fibrous tissue inside the cavity and peribronchial thickening remain. This patient had been given 16 bronchoscopic treatments by Dr. William Frederick Moore, at the Bronchoscopic Clinic, Department for Disease of the Chest, Jefferson Hospital.

bronchoscope attached to the motor aspirator removes the secretions as fast as they are encountered on the way down. Then the protected-eye aspirating tube is introduced through the bronchoscope and inserted deeply into the fistula, and the pus aspirated. If more than one fistula is encountered, each is taken in turn. The independent aspirating tube is then withdrawn and a fresh one is introduced, having a short piece of rubber tubing attached to its proximal end. To this rubber tube, a graduated glass syringe, containing the fluid to be injected, is attached. The whole procedure ordinarily requires 3 or 4 minutes. In children, no anesthetic, general or local, is used. In adults, a little cocain is applied to the region of the superior laryngeal nerve in the laryngopharynx. In many adult patients, even this application of cocain is omitted after the first few treatments.

Vaccine therapy is of much increased efficiency when carried out with culture material obtained bronchoscopically from the suppurative focus itself, without contamination with the secretions of the mouth, which contain such a bountiful bacterial flora as to render it difficult for the laboratory technician to isolate the organisms predominant in the pulmonary suppurative focus. In hundreds of cases at the Bronchoscopic Clinic, vaccines prepared from uncontaminated specimens removed bronchoscopically from the pulmonary lesions have produced a higher percentage of beneficial results than were ever obtained from sputum.

General Treatment. It would be a serious mistake to rely solely on bronchoscopic curative measures to the neglect of rest in bed outdoors, postural drainage, vaccine therapy, diet, and general therapeutic measures. These remarks seem platitudinous; but if the bronchoscopist is not eternally vigilant, he will find the internist and particularly the patient relying more and more on the local bronchoscopic drainage, lavage and medication, to the neglect of other measures. One of the most urgent needs of vigilance is to see that as the patient begins to improve, he does not use up the gain by increased work or exercise. It must be strongly impressed upon him that he must build up this increase into a reserve, and not fritter it away in either work or pleasure. After drainage has been improved up to a good degree of efficiency, it is often well to send the patient to a suitable climate. This is especially useful in winter. If the patient can go for a few months to a warm sunny climate, where he can rest outdoors with pleasure and comfort, the interruption of the treatment is often well worth while. Of course, it is true that the stimulus of a cold, sunny climate is sometimes beneficial; yet

the discomfort is so great, that in spite of all that can be done, the patient will not rest in bed outdoors the required number of hours; and the hours of sunshine are fewer in most cool localities. These, however, are matters for decision by the internist.

It should be repeated, for emphasis, that the successful treatment of a case of pulmonary abscess requires close cooperation of the bronchoscopist, the internist, the roentgenologist and the surgeon, under the general supervision of the internist.

BRONCHOSCOPY IN BRONCHIECTASIS.

The bronchoscope has proven exceedingly useful in the diagnosis and treatment of bronchiectasis.

Diagnosis. The diagnosis in a well marked case is easily made by noting the increased diameter of the bronchi, the dilations alternating with contractions, the walls chronically inflamed, possibly eroded or ulcerated, and smeared with foul pus. If any doubt exists, lung mapping by a stereoroentgenogram after bronchoscopic insufflation of powdered subcarbonat of bismuth, as explained in a preceding paragraph, will determine the diagnosis. One of the most important diagnostic points in which bronchoscopy is invaluable is the determination of the presence of neoplasms, foreign bodies, compressive or cicatricial stenoses, which may exist in the absence of bronchiectasis or coexist with it.

Prognosis. As with abscess, it is well to remind the patient and the practitioner that bronchiectasis is essentially a very chronic disease, which under any method of treatment, climatic, medical, surgical, postural, et cetera is slow in progress toward a cure. The thoracic surgeon, after operation, expects to treat the patient for months. Viewed from this standpoint, bronchoscopic treatment, when added to a proper regimen, carried out under the supervision of the internist, is the most promising method of treatment known today. Cured patients, in some instances, occasionally cough up a crust or a small mass of mucopurulent secretion. This comes from the area in which cicatricial tissue and epithelium devoid of cilia has replaced the normal ciliated epithelium destroyed by prolonged suppuration. Necessarily, the drainage is imperfect from such areas, and consequently the secretions accumulate until sufficient in quantity to be expelled by cough; but the symptoms of the disease in these cases have disappeared.

Treatment. The first indication is to dilate any stricture that may exist, and to remove any other obstruction to drainage, such

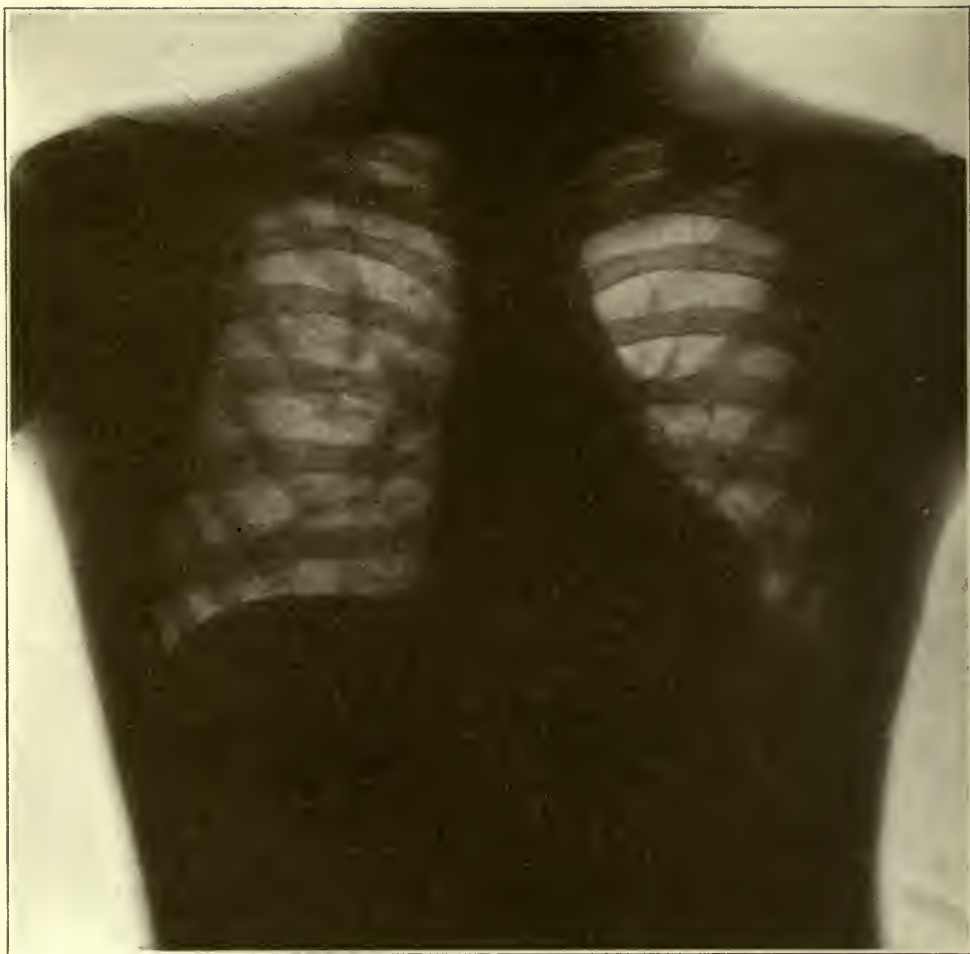


Fig. 8. Roentgenogram of boy, aged $12\frac{1}{2}$ years. Free expectoration of pus following an attack of pneumonia $2\frac{1}{2}$ years before admission. Ray study shows chronic inflammatory process in lower posterior portion of left chest, adhesions of left diaphragm and retraction of heart to left. Evidence of dilated bronchi or small cavities behind the heart. Bronchiectatic areas visible.



Fig. 9. Same patient as in Fig. 8, taken six months later. Bronchoscopic aspiration and endobronchial medication were carried out without anesthesia, by Dr. Gabriel Tucker, at the Bronchoscopic Clinic, University Hospital. Roentgen ray study shows decrease of density posterior to heart and less evidence of peribronchial thickening along the left border of heart; diaphragms seem equal on both sides. Clinically very little pus was being coughed up at this time, and patient had gained 20 lbs. in weight. This patient made a complete subjective and objective recovery under bronchoscopic treatment by Dr. Tucker.

as granulations or granulomata. In the so-called idiopathic types, considerable benefit has resulted from the endobronchial lavage and endobronchial oily injections mentioned under lung abscess, care being taken not to flood healthy tissue. Our best results at the Bronchoscopic Clinic have come from aspiration of pus, wiping the diseased area, and swabbing with one of the preparations mentioned under Pulmonary Abscess. In some cases, the insufflations once weekly of about 20 grains of bismuth subcarbonat have been useful. It is probable that if bronchoscopic study were carried out in every case, definite causes for many so-called "idiopathic" cases would be discovered, and curative treatment would follow. Vaccine therapy is most efficient when the vaccines are prepared from material bronchoscopically removed, uncontaminated with oral organisms.

BRONCHOSCOPY IN BRONCHOLITHIASIS.

Lung stones may belong to one of three classes: (1) true calculi; (2) calcareous deposits in the tissues that sloughed loose and gained entrance into the bronchi; or (3) pneumoconiotic, silicotic, or anthracotic material that has loosened by disintegration or suppurative liquefaction of the encasing tissues. In a case of bronchiectasis that came to the Bronchoscopic Clinic, the bronchoscope assisted in removal of a true calculus together with pneumoconiotic material (Fig. 12). The endogenous origin of the calculus, or true lung stone, and the exogenous origin of the gritty material were shown by the chemical and physical examination of Prof. Phillip B. Hawke and his associates, Dr. Bergeim and Dr. Smith. Removal of the broncholithic material did not greatly or permanently improve the bronchiectatic symptoms, which had been present for many years. Notwithstanding the failure to improve the bronchiectatic condition in this case, it seems reasonable to assume that the removal of the irritating broncholithic material saved the patient from troubles he would have had from its presence. Anyway, it would seem that bronchoscopy for the prompt ridding of the bronchi of the inevitably irritating lung stones is indicated in every case suspected of having either a calculus or calcareous or pneumoconiotic material in the bronchi. The two diagnostic means leading to suspicion of the presence of these conditions are the roentgen ray and the observation of gritty material in the sputum. In the case mentioned (Fig. 12) the stone showed plainly in the roentgenogram made by Dr. David R. Bowen, who made the



Fig. 10. Illustration showing lung mapping by the author's method of bronchoscopic insufflation of dry powdered bismuth subcarbonat. In this instance the insufflation was done for localization of the bronchi relative to the penetrating projectile.

diagnosis, and who found the calcareous shadow gone after the bronchoscopy.

CHRONIC BRONCHITIS.

Old, intractable cases of purulent bronchitis which have failed of cure by general means including hygiene, change of climate, internal medicine, and inhalations, have many times been cured by local bronchoscopic aspiration and medication, combined with vaccine therapy, the vaccines being prepared from swab specimens removed with a careful technic, planned to avoid oral contamination. The local measures used consisted in aspiration of secretions and the application with a bronchoscopic atomizer of solutions of monochlorophenol, half of one per cent in liquid petrolatum. In some cases, applications on a swab were found more efficacious. The medicaments used were argentic nitrat in ten per cent solution; argyrol in twenty per cent solution; zinc chlorid in one tenth of one per cent solution. In recent years, the endobronchial injection of ten c.c. gomenol, twenty per cent

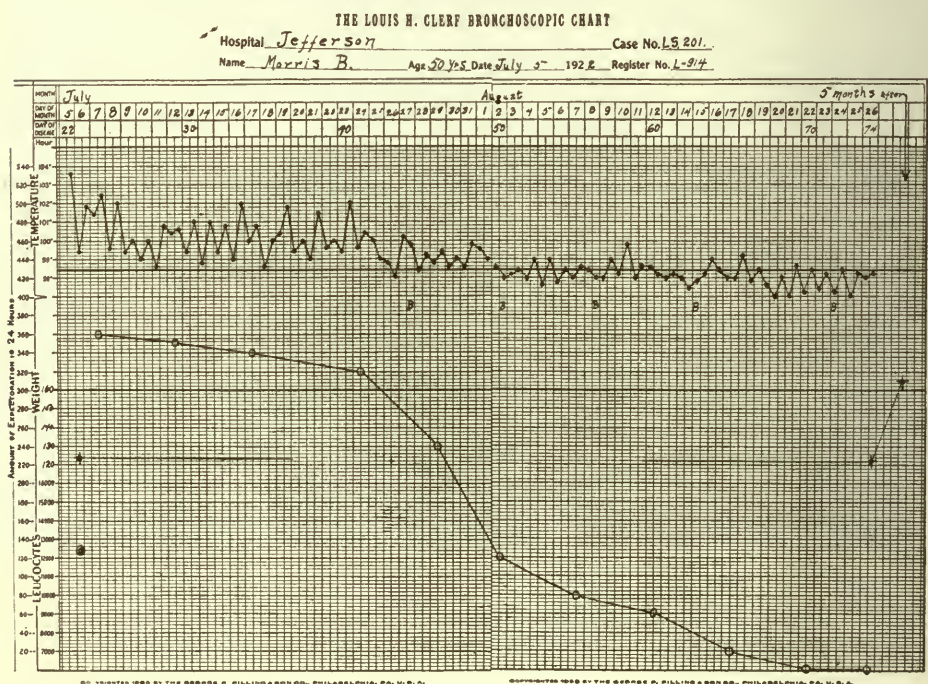


Fig. 11. The Louis H. Clerf chart for determining the trend of the patient with pulmonary suppuration. This chart shows a favorable trend. Had the trend been unfavorable, the treatment would have been discontinued.

solution in liquid petrolatum, has been used with very good results. For calling our attention to this preparation, we are indebted to Jean Guisez.

In the treatment of pulmonary conditions at the Bronchoscopic Clinic, it has seemed to us better not to flood the healthy lung with solutions of any kind, but to limit the medicinal or cleansing applications to the diseased areas.

In all chronic suppurative pulmonary diseases, it should be remembered that cilia have been destroyed. Hence ciliary drainage is impaired, and this impairment renders drainage especially bad in the upright posture, because the resistance of gravity has to be overcome. For this reason, all cases of chronic pulmonary suppuration of any kind should not be in the erect posture for more than four hours daily, and these hours should be at scattered



Fig. 12. A, a broncholith from bronchiectatic purulent accumulation removed by peroral bronchoscopy under local anesthesia, in case of a man aged 20 years, with long standing bronchiectasis. B, inorganic material from the same pus. The nucleus of the broncholith, A, was of similar character to that of the inorganic material, B.

intervals through the day, not continuous. Rest in bed outdoors in a sunny, dust free location is the most favorable environment for recovery. Diet should, of course, be regulated, and free elimination should be maintained. Vaccine therapy is of greater efficiency when the vaccines are prepared from material bronchoscopically removed, without oral contamination.

BRONCHOSCOPY IN TUBERCULOSIS.

Diagnosis. Obviously bronchoscopy is not called for in the routine diagnosis of pulmonary tuberculosis. There are, however, many cases of suspected intrathoracic tuberculosis in which bronchoscopy is of the utmost value. In any case of suspected tuberculosis with sputum that is abundant and purulent, and yet constantly free from the bacillus tuberculosis, bronchoscopy is indicated for diagnosis. Tuberculous cases are often seen with scanty sputum in which no bacilli can be demonstrated, and in such cases bronchoscopy is rarely indicated; but if there is an active suppurative lesion, the absence of bacilli should raise at once a diagnostic question; and in the solution of this question, the bronchoscope comes second only to the Roentgen ray examination.

In 164 such cases at the Bronchoscopic Clinics, 52 were found to be tuberculous; of the other 112 there were 14 that did not continue attendance until the diagnosis was completed. The remaining 98 proved to be nontuberculous. Of these, 54 were due to foreign body, and 44 were nontuberculous abscesses, bronchitis, bronchiectasis, glandular suppurations, lues, etc. In these diagnoses, the bronchoscope played an important though not exclusive part, full advantage being taken of all other diagnostic means.

Bronchoscopic Appearances in Tuberculosis. The subglottic infiltrations from extensions of laryngeal disease are usually of edematous appearance, though they are much more firm than in ordinary inflammatory edema. Tracheal ulcerations are rare, except as direct extensions of ulceration of the larynx. The trachea is relatively rarely involved in tuberculosis, but we may have in rare cases the pale swelling of the early stage of perichondritis, or the later ulceration and all the phenomena following the mixed pyogenic infections. These same conditions may exist in the bronchi. In a number of instances, the entire lumen of the bronchus was occluded by cheesy pus and debris of a peribronchial gland which had eroded through. As a rule, the mucosa of tuberculosis is pale, and the pallor is accentuated by the rather bluish streak of vessels, where these are visible. Erosion through of peribronchial or peritracheal lymph masses may be associated with granulation tissue, usually of pale color, but occasionally reddish, and sometimes oozing of blood is noticed. A most common picture in tuberculosis is a broadening of the carina, which may be so marked as to obliterate the carina and to bulge inward, producing deformed lumina in both bronchi. Sometimes the lumina are crescentic, the concavity of the crescent being internal, that is, toward the median line. Absence of the normal anterior and downward movement of the carina on deep inspiration is almost pathognomonic of a mass at the bifurcation, and such a mass is usually tuberculous, though it may be malignant, and, very rarely, luetic. The only lesion visible in a tuberculous case may be cicatrices from healed processes. In a number of cases there has been a discharge of pus coming from the upper lobe bronchus.

Bronchoscopic Treatment of Tuberculosis. In most cases of pulmonary tuberculosis, there is no indication for bronchoscopic treatment; occasionally, however, the bronchoscope is very urgently indicated. When the physical signs and the Roentgen ray indicate bronchial obstruction, bronchoscopy should be done to determine the character of the obstruction. If due to cheesy

masses, granulations, and inflammatory products, such cases are quickly relieved, often at a single bronchoscopy. If there is a fibrous or a compressive stenosis, the dilator can be used without harm and with benefit. Not infrequently, cases are sent in for bronchoscopy with the history of cycles of chilliness, fever, purulent expectoration alternating with quiescent symptomless intervals, the ray showing accumulation of pus in the interval and its discharge during the febrile period. These cases have, in every instance, been relieved by improving the drainage by bronchoscopic means. A number of the earlier cases have since recovered and their lesions have cicatrized.

BRONCHIAL ASTHMA.

This has been studied at the Bronchoscopic Clinic as cases were referred to us. The cases associated with chronic bronchitis have been found to be markedly benefitted in fully half of the cases, and a number of cures have been obtained. The cases unassociated with chronic bronchitis have not been benefitted. In a number of instances, a benign growth has been found, and its removal has resulted in a cure. The foregoing is the result of our general observations. Dr. William F. Moore, Bronchoscopist to the Asthma Clinic, Jefferson Hospital, has been making special studies in asthmatic cases, and will embody his results in a full report.

NEOPLASMS OF THE LUNG.

Malignant and benign growths of the lung are not within the scope of the present paper; but two findings should be stated here:

1. Malignant disease of the lung has been found bronchoscopically in many cases of abscess in which the malignant nature of the process was unsuspected⁷.

2. Benign growths are occasionally the unsuspected cause of lung suppuration. Benign neoplasms causing suppuration have been found bronchoscopically at the Bronchoscopic Clinic, excluding syphilomata, tuberculomata and granulomata, the latter probably secondary to suppurative processes. The benign growths included endothelioma, fibroma and chondroma. The patient from whom the endothelioma was removed is still alive and well at the end of seven years, so it seems that we must consider the growth clinically benign though histologically malignant.

CONCLUSIONS.

Ultimate conclusions in medicine are never possible because of never ceasing progress. Conclusions at any particular time can present only the status of the moment. In such a group of varied pathologic conditions as are found in lung suppuration, for definite conclusions on all phases of the subject of bronchoscopic diagnosis and treatment, complete data on at least ten thousand cases should be available for analysis. Our experience, so far, at the Bronchoscopic Clinic has, however, definitely established beyond question the following facts:

1. Bronchoscopy for diagnosis is of great assistance in obscure cases, and has often revised the previous clinical diagnosis from tuberculosis to nontuberculous suppuration; from abscess to bronchiectasis; from abscess, bronchiectasis, tuberculosis, et cetera to malignant growth; from pulmonary hemorrhage to rupture of dilated tracheal vessels. We have had many cases showing bronchoscopic revision of the diagnosis in the reverse order of each of the foregoing citations.

2. Bronchoscopic treatment of endobronchial malignancy was attempted only once, and was done with the intention of taking a specimen for diagnosis. The growth was removed entire and was found to be an endothelioma. Doubtless in malignant disease of the lung, bronchoscopy will be useful only diagnostically, though the direct application of radium is feasible mechanically.

3. Bronchoscopic treatment of tuberculosis will doubtless be limited to cases of periodically obstructed drainage of pus accumulations.

4. In all nontuberculous, nonmalignant, not hopeless cases of lung suppuration, peroral bronchoscopic drainage is worthy of first consideration. Alone or combined with endobronchial bronchoscopic medication, it will benefit most cases and will afford absolute cures in a certain percentage of cases.

5. In a number of cases, not of long standing, the aspiration incident to a diagnostic bronchoscopy has brought the temperature to normal and started the patient on the road to recovery.

6. It would, however, be a most unfortunate error if bronchoscopy, for either diagnosis or treatment, were undertaken by anyone as an independent procedure. These procedures can accomplish their greatest usefulness only when carried out under the observation and guidance of the internist, whose broad viewpoint will determine the suitability of the case, by eliminating those in which external surgery or medical care alone is needed.

7. A number of brilliant results have come in those cases in which the internist has enabled the patient to hold his own, but without any progress toward cure. In these cases, the addition of biweekly bronchoscopic drainage has proven the little addition that has turned the tide in the patient's favor, and thus contributed to a cure.

8. The bronchoscopic means of improving drainage are in the removal of obstructing granulation tissue, the dilatation of stenosed bronchi, and restoration of ciliary action by aspiration of areas of purulent drowned lung.

9. Thus it is seen that the bronchoscopic treatment of lung suppuration is, in principle, not new, but very old; dating back to the predecessors of Hippocrates, who preached and practiced drainage of suppurating foci. The only new phase is the development of the instruments and technic to the point where the lung can be drained by the inserting of the bronchoscope through the mouth into the suppurating lung harmlessly, painlessly, without anesthesia, in a minute's time, without hospitalization of the patient.

10. As a rule treatment should be begun as early in the case as possible (Moore). In some instances, however, a cure has resulted from bronchoscopic treatment of cases of many years standing; almost a whole lifetime of over twenty years in two instances.

11. In answer to the frequently asked question, "What cases are suitable for bronchoscopic treatment?" the present state of our knowledge enables us to say: "Any case, not hopeless, in which the internist feels the need of supplementing medical care and treatment with bronchoscopic drainage through the mouth, and any case, not hopeless, in which the surgeon feels that external operation is for any reason inadvisable for the time being."

REFERENCES.

1. Lukens, Robert M., Moore, W. F., and Funk, E. H. *Bronchoscopy for Treatment of Pulmonary Abscess*. Med. Clinics of North Amer., January, 1923. Wm. B. Saunders Co.
2. Lukens, Robert M., and Moore, W. F. *Lung Suppuration; Its Treatment by Bronchoscopy*. Atlantic Medical Monthly, 1924.
3. Jackson, Chevalier. *The Bronchial Tree, Its Study by Insufflation of Opaque Substances in the Living*. Amer. Journ. Roentgenol., 1918, v. 5, p. 454.
4. Lynah, H. L., and Stewart, W. H. *Roentgenographic Studies of Bronchiectasis and Lung Abscess of Direct Injection of Bismuth Mixture through the Bronchoscope*. Amer. Journ. Roentgenol., 1921, v. 8, p. 49.

5. Fetterolf, George. The Paratonsillar Tissues in Relation to Posttonsillectomy Abscess. *Proceedings Am. Laryngol. Soc.*, 1923.
6. Moore, Wm. F. Pulmonary Abscess. *Journ. Amer. Med. Assn.*, Apr. 29, 1922.
7. Jackson, Chevalier. Endothelioma of the Right Bronchus Removed by Peroral Bronchoscopy. *Am. Journ. of the Med. Sciences*, March, 1917, No. 3, vol. 152, p. 371.
8. Jackson, Chevalier. Caisson Bronchoscopy. *Surg., Gyn., and Obst.*, Oct., 1917, p. 424.
9. Jackson, Chevalier. Observations on the Pathology of Foreign Bodies in the Air and Food Passages. Analysis of 628 Cases. *Mütter Lecture*, 1917. *Surg. Gyn. and Obstet.*, March, 1919, pp. 201-261.
10. Jackson, Chevalier. *Bronchoscopy and Esophagoscopy*. Textbook, 1923, W. B. Saunders and Co., Philadelphia.
11. *Peroral Endoscopy and Laryngeal Surgery*. Textbook, 1914.

DISCUSSION.

DR. R. M. LUKENS, Philadelphia, Pa.: The object of the irrigating apparatus, as used in the Jefferson Clinic for irrigating these lesions, is to keep the solution at a uniform temperature and at uniform pressure. The temperature of the solution in the "thermo-reservoir" is 115° F., at the tube 105° F., entering the lung at about 100° F. This apparatus was shown in the picture. It consists of a 1,000 c.c. steel thermos bottle, with a gauge at the side to show the fluid level. At the bottom is a stopcock, which controls the flow of the fluid, and at this valve (indicating), the fluid is allowed to drain out after irrigation of the abscess cavity, the cold being run off. The hot fluid is run in fresh, so that there is no cooling of the fluid in the tube. A thermometer is connected into the delivery tube to show the temperature of fluid just before entering the lung. The two-way irrigating tube is composed of a small irrigating pipe and a larger aspirating pipe. It is just a slight modification of Dr. Yankauer's irrigator to suit conditions at our clinic. The solution goes in, washes around, and is drawn off immediately.

Just recently, in order to irrigate the lower lobe, we have been using a small caliber tube thru the Jackson aspirating bronchoscope. This tube often becomes bent and obstructs the view, so that we have devised a channel inside the irrigating bronchoscope thru which the irrigating tube can be guided. This tube is run right down into the bronchus and the irrigation started. After granulations around the orifice of the bronchus have been removed, very often there is some stenosis of the orifice, and for that we use the regular Jackson bronchial dilator. This is inserted into the narrowed bronchus, opened and drawn out.

I wish to say just a word about team work. In our clinic, every man not only does the operating but also assists. The nurse is especially trained to care for the instruments and to act as third assistant, and when we have to use a new pupil nurse, we have her begin two or three weeks before the old nurse leaves. In this way the work runs very smoothly, and the patients do not object to the treatment. The place to do bronchoscopy is at a hospital especially equipped. We feel that it is not wise to run from one place to another for this work, but that it should be done where everything is at hand, and where there is no cause for hurry or uncertainty.

DR. W. F. MOORE, Philadelphia, Pa.: We are very much interested at the Bronchoscopic Clinic in tracheobronchial asthma, the treatment of which Dr. Jackson has shown in some of the pictures. These cases have been carefully tried out in the asthma clinic as to sensitization, and we feel that in some cases the symptoms are due to local conditions within the lungs themselves, in the bronchi. In treating these cases, we have been using a solution containing 20 mms. of (1/1000 solution) adrenalin and 10 mm. of 10 per cent cocain in a dram of normal saline solution. This has been injected directly into the right and left bronchi. We have treated sixteen cases during the last four or five months, and their progress has been fairly satisfactory. I suspect, that after we have treated a large number of cases, we will decide that the question of the, at times, accompanying secondary symptoms, chief among which is emphysema, will have a great part to play in their cure, or the alleviation of their symptoms.

DR. ELMER H. FUNK, Philadelphia, Pa. (by invitation): What I have to say in this discussion is from the point of view of an internist who has had the privilege of seeing many of the patients before, during, and after bronchoscopic study and drainage. Internists are finding more cases of pulmonary suppuration than formerly. Whether this is due to an increased incidence or more frequent recognition, or both, is not for discussion this morning. The important thing is, that in bronchoscopic drainage as practiced by Dr. Jackson and his assistants, we have a new and valuable procedure in the treatment of localized lung suppuration. Until recently, when such a patient came to us we debated whether or not to resort to surgery, realizing the high mortality of operative procedures. Lockwood states that the literature contains reports of 1,117 cases of lung abscess, with an average mortality with surgical treatment of 34.6 per cent. Lilienthal, who may be regarded as having had an extensive experience with resection of the lung for abscess, had a mortality of 54 per cent. In bronchiectasis, Graham found, among 48 reported cases submitted to lobectomy, an operative mortality of 52 per cent. This does not mean that all of those who recovered from operation were cured; many of them no doubt were unimproved. Is it any wonder that internists have been loath to refer their patients for such a formidable operative procedure? We who were conservative waited, hoping that drainage by way of the bronchial tubes would become established in due time; and when this occurred, we endeavored to aid drainage by posture of patient. Some have tried to help matters by inducing pneumothorax. Rich and others have reported favorable results. This method, however, is not without danger, and two deaths from this procedure have been reported by Wessler.

When we review the brilliant foreign body work of Chevalier Jackson, and note how well his patients with lung suppuration cleared up after the causative foreign body was removed by bronchoscopy and the establishment of adequate drainage, the conclusion is inevitable that a trial of the same procedure has a rational basis in those cases of lung suppuration not due to foreign body. Furthermore, the value of any procedure is enhanced when it can be shown that it is safe. From my observation of the work done by the bronchoscopists at Jefferson, I can affirm its safety in their hands. There is no mortality and no appreciable ill effects from

bronchoscopy in suitable cases. It is not fair to expect the method to be of value in moribund patients, or those with widespread suppuration or multiple abscess formation, or with complications such as empyema, etc. On the other hand, to witness the reduction in the amount and fetor of the sputum, the improvement in the cough, the improvement in the general condition as a result of the lessened toxemia in patients treated bronchoscopically, is to witness that which occurs less commonly with other present day methods. In one of the patients reported by Dr. Jackson's assistants, Dr. Lukens and Dr. Moore—a young woman with a chronic abscess of years' duration—bronchoscopic drainage was followed by such striking benefit, that she was able to return to the society from which she withdrew because of the offensiveness of the cough, expectoration, and fetor of the breath. In this same patient, the trophic improvement was shown in a striking way in the improvement in the curved finger nails. Shortly after treatments were begun, a ridge occurred at the roots of the nails. This was watched, and it was found that the incoming nail was more normal in shape and appearance than the outgoing nail beyond the ridge.

I grant that there are certain cases of lung suppuration which seem to clear up without treatment. Others respond to postural drainage, therapeutic pneumothorax, thoracotomy, and lobectomy. Personally, if I had a localized lung suppuration, I would prefer that my physician should call in as an aid in treatment the services of a skilled bronchoscopist. And I would have a confidence in the help which bronchoscopy for drainage can give. It will not work in every case, but in the hands of skilled men its safety commends it to trial. And I know it has helped a number of patients, in whom radical surgical procedures were contemplated, to evade the knife and recover.

DR. J. M. WAUGH, Cleveland, Ohio: I would like to ask two questions: First, the children shown on the screen all looked well nourished, and I would like to know what was the particular cause for these abscesses in the children? Were they postpneumonic, postinfluenzal, or due to something else? I believe children show abscesses from these causes more frequently than adults.

I would like to ask Dr. Moore about the pathology which he said was found in the cases. Will he please say a word as to the clinical appearance in the bronchi as seen through the bronchoscope, which he thinks is characteristic of asthma? I would also like to know how frequently these treatments are carried out.

DR. CHEVALIER JACKSON, Philadelphia, Pa. (closing): I am unable to answer Dr. Waugh's question as to whether one cause is more prevalent than another in children. I promised not to say anything about foreign bodies, so I hesitate to say anything about the peanut, which is the commonest cause. As to the frequency of treatment, I think Dr. Moore and Dr. Lukens had better give their view of that.

I think that mechanics who do bronchoscopy need a balance wheel. We need to consult with the roentgenologist, with the surgeon, and with the internist; and when it comes to the final decision about this method, we should ask the internist, for he is the man who is the balance wheel. With his deductive reasoning and his broad viewpoint, he is the man to ask whether bronchoscopy is indicated in any particular case.

SOME PRACTICAL POINTS IN THE PROGRESS OF MASTOID SURGERY.

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Mastoid surgery has, during the past two or three decades, progressed as rapidly if not more so than general surgery. Mastoidectomy is no longer an operation much dreaded by both patient and surgeon, and attended by a high mortality rate, serious complications and sequelae, long and painful convalescence, unsightly deformities; protracted otorrhea and impairment of hearing. I do not mean to say that all of these disagreeable factors have been removed, but certainly they have been diminished to a great extent. Many of us have had occasion from time to time to feel dissatisfied with our mastoid work. It was thought that much benefit might be derived from an exchange of ideas and general discussion by the members of this section, of some of the practical points leading to a more uniform solution of the causes of many unsatisfactory results. Our aim is perfection but our progress is slow.

The diagnosis of an acute mastoiditis is as a rule easy. It is in the complicated and atypical cases that we have to utilize all the known diagnostic measures at our command. It ought to be seldom indeed, that we should feel the necessity for opening the mastoid process for purposes of exploration. Many of you have had, especially in recent years, cases of so-called mastoidalgia, with subjective symptoms of mastoiditis, such as pain in the mastoid region, and pain on pressure over the mastoid process. We are also encountering in increasing numbers mastoid infections without a suppurative otitis media. Dr. Horace Bonner and I, in 1917, reported a case of what we thought to be idiopathic mastoiditis, but close observations since that time have convinced me that the infection always passes through the middle ear. In such cases, a leucocyte count of fifteen thousand and over would suggest an infection, but a normal count would practically rule out a mastoiditis. The routine use of the laboratory is absolutely essential.

The Roentgen rays have become an indispensable adjunct in the differential diagnosis in external and middle ear infections. It is also valuable in portraying certain anatomic relationships, and

the degree and extent of pathology within the mastoid process. Much emphasis must be placed upon stereoscopic pictures or plates. Not only should one process be studied stereoscopically, but both should be placed side by side to facilitate a most minute comparative study. I wish to suggest to those men who have limited X-ray service, to cooperate and study with your radiologists this most important point. It may be necessary, at least it is helpful and desirable, that you acquire the ability to interpret the skiagraphs yourself.

The incision should be from three to five millimeters posterior to the junction of the auricle with the scalp, and should conform to its degree of curvature. Above, it should extend forward to a vertical line drawn through the tragus, and below, over the center of the mastoid process and downward sufficiently to give a good exposure of the tip. A curvilinear incision such as this, when healed, is hidden from view, and is a great improvement over the former more posterior and nearly vertical one.

It should be our object in all surgical work, and this is especially true in destructive or excavative bone surgery, to provide for regeneration as far as possible. Nothing aids more in the formation of new bone than the periosteum. I therefore wish to make this plea for its careful preservation. It should be carefully separated, elevated and retracted, and upon completion of the operation, its cut edges brought together and sewed with a catgut suture. At least two thirds of the skin wound is closed with interrupted silkworm gut sutures at the time of the operation. Usually the lower third is left open, through which the packing or drainage tube protrudes, and subsequent dressings are made. Union is often by first intention, with a minimum of scar tissue and a short period of convalescence. There are a few good aural surgeons who still adhere to the open method of treating postoperative wounds. Personally, I cannot see the necessity for the open method in more than from three to five per cent of cases.

Another practical point is gentleness in dealing with the soft parts. Do not maul and unnecessarily traumatize them. Elevate gently the soft parts of the external auditory canal, especially that portion covering the inner third. The delicate epithelium is easily destroyed. It is most necessary in the process of repair.

It is the duty of every surgeon working in this field, as well as in nasal accessory sinus work, to remove as far as possible all pathology. All diseased mastoid cells and contiguous infected bone and diploe should be removed down to the inner plate of

the skull, if we hope to get a complete and quick convalescence. The cells in the tip of the process should be eliminated as far as possible, but the tip need not be removed. Dench advises the removal of the entire tip, because he has had it become a sequestrum requiring subsequent removal. I have never had one become necrotic. The exenteration I believe is best accomplished with a mallet, gouges, chisels and curettes, together with a good rongeur forceps. Some very good men use burrs driven by an electrical engine. I am not at all familiar with the burr. My early experience in mastoid surgery was with the chisel and curette, and naturally I prefer to adhere to this technic. I do believe, however, that it is possible to do better individual work and with a greater factor of safety with the curette than with the burr.

The exenterated cavity in a complete mastoidectomy very often presents, especially in children, an inverted cone resting upon a triangular base. The apex of the cone corresponds with the antrum, or a point along the aditus at antrum, near the attic. The three angles of the triangular base are located as follows: One pointing downward, and corresponding with the tip of the mastoid process; one extending backward toward the occiput, and corresponding with the backward extension of the mastoid cells and usually overlying the lateral sinus; and one extending forward through the temporal bone and very often into the root of the zygoma. In every case, the posterior bony wall of the external auditory canal should be removed almost as far down as in doing a radical operation. Many operators leave a deep hollowed out cavity with vertical and overhanging edges, hoping that Nature will fill in this gourd like space. This practice should receive our earnest condemnation, because in many instances the space persists or becomes filled with cholesteatoma. These edges and the overhanging outer table of the skull should be removed and curetted smoothly, the object being to get a smooth, bevelled bony cavity to come in contact with the under surface of the soft parts at all points if possible. In bevelling the floor and the edges of your cavity, this space, if your periosteum has been preserved, can be permanently eliminated by the formation of fibrous tissue and new bone, with a minimum of deformity and without the instillation of a foreign substance, such as bismuth paste or a blood clot, after the method of Reik.¹

We now come to what I believe is one of the most important points in mastoid surgery, viz., our ability to prevent a secondary infection. Nothing should please one more than to be able to

say to the patient after a mastoidectomy, that he is reasonably sure that the infection cannot recur in the operated mastoid. This statement you cannot safely make, unless you have almost, if not completely, obliterated the only avenue of communication with the outside world—the aditus ad antrum, through which all infections entering the mastoid region must pass. During the last three and one-half years, in a series of forty-one mastoidectomies, I have had three secondary infections among my own patients requiring a second operation. During the same period, I have seen and operated upon several cases coming from my colleagues. In all of these, we found the mastoid cavity only partially filled either with bone, or fibrous tissue and cholesteatoma, and with a patent aditus leading directly into the middle ear. Many aural surgeons have for years been enlarging the aditus and removing its overlying bony covering, and obliterating it entirely to within a few millimeters of the attic, in such a way that in the healing process the mastoid cavity was entirely shut off from the tympanic cavity. It only remained for Prof. Bárány² to emphasize and point out the great practicability of this procedure. He goes much farther, and advises his new radical operation for all cases in which the middle ear is chronically diseased. He states that the aditus can be closed by fibrous tissue and epithelium growing from the tympanic cavity posteriorly through or into the aditus, effecting its permanent closure. Professor Bárány's suggestions last year before this section were practical and full of merit, and should receive our careful consideration. If his new radical operation, possibly with some modifications, is perfected in this country, it will aid us in obtaining dry ears without the apparent necessity, at least, of the elaborate, delicate and many times unsuccessful technic of plastic flaps and skin grafting. If this closure can be brought about from before backward in radical mastoid operations, I am convinced that it can be and has been closed in many of our cases from behind forward without any special effort upon our part to bring it about. I wish further to state, that if the overlying bone is removed sufficiently and the contour of the aditus made flat, so that the periosteum and soft tissue can come in contact with its floor, it will be obliterated in practically every case, and the mastoid cavity permanently separated from the middle ear, thereby precluding the possibility of a secondary infection. The curetting must always be done from below upward. There is one point about which I must warn you, especially in children, and that is, to approach the attic very care-

fully so as not to destroy the ossicles. This occurred in one of my cases.

It has not been very long ago that we were apprehensive if the lateral sinus or dura happened to be exposed during an operation. I have never had any bad after effects that could be traced to their exposure. It is my sincere desire, that if any member or members present have had ill effects from this cause, they will report them. Obviously these exposures, intentionally or otherwise, very often aid us in making a diagnosis of sinus involvement, extradural abscess, etc., and to deal with them promptly, which could only have been done by subsequent exploration. It has been my opportunity in several cases upon the exposure of the sinus to find a perisinus abscess, or to find it entirely collapsed, or to be filled with a well organized thrombus. A fatal issue is very often avoided by prompt and rational surgical treatment of such grave complications. Pages could be written upon the diagnosis and treatment of sinus thrombus, but time will permit me to mention briefly only a few of the cardinal points. The onset is usually characterized by a chill followed by a sharp rise in temperature, the so-called "church steeple" temperature chart. One of our greatest diagnostic aids is frequent and repeated blood cultures, taken before the chill. With a positive blood culture and other confirmatory symptoms, one is justified in resorting to further surgical procedures.

We are fortunate today in having blood transfusion at our command. Whole blood seems to be preferable, because of its more efficient bactericidal properties, after the method of Unger. There seems to be a divergence of opinion and lack of proof from a bactericidal standpoint, but be that as it may, new blood is of decided value in tiding a patient over a period when he has used up all of his own resistance.

A close study of the histories of many cases, over a period of more than fourteen years, has convinced me that most of our acute intracranial complications take place along with, or soon after the infection invades the middle ear. In acute infections of the tympanum, the entire mucous membrane is markedly engorged, with the infection travelling rapidly in all directions, especially backward through the aditus into the antrum, and then by several direct routes, viz., through the emissary veins and lymphatics into the cranial cavity, and even into the brain itself. Very often symptoms suggesting intracranial complications are present from the outset, regardless of how early and how freely the drum membrane is incised. Most all of these

complications, except meningitis, if promptly diagnosed can be very often satisfactorily dealt with surgically.

It has been correctly stated that a copious purulent discharge almost always means mastoid involvement. The area of the tympanic cavity is small, and is, therefore, considered incapable of secreting pus in such large amounts. Many such cases, especially in children, should be operated upon, first, to terminate the infection and, second, to preserve the hearing. It should be the exception now-a-days to get impairment of hearing following an operation for acute mastoiditis.

I have endeavored to stress a few points in the progress of mastoid surgery which I am confident are responsible for our present day results as compared with those of former years, and which I believe, if correlated and put into broad general use, will be the means of still further advancement.

CONCLUSIONS.

1. The advancement in laboratory methods and the perfection of the stereoscope in the study of X-ray plates are of great service in the diagnosis of mastoiditis.

2. Location, shape and prompt closure of at least two-thirds of the incision gives the best cosmetic result.

3. Preservation and replacement of the periosteum is necessary for bone regeneration.

4. Protection of epithelial membrane adjacent to and within the tympanum necessary in epidermization.

5. Thoroughness in removing all pathology.

6. Bevelled bony cavity of great value in the permanent obliteration of the exenterated mastoid process, without the introduction of a foreign substance.

7. Obliteration of the aditus and its permanent closure effected by the regeneration of new bone and fibrous tissue, thereby preventing secondary mastoid infections.

8. Explore the dura and lateral sinus at the time of operation, if you have any reason to feel that they might be involved.

9. A close study of histories and subsequent observations strongly suggest that acute intracranial complications take place very often simultaneously with the middle ear involvement.

10. Chills, church steeple temperature chart and positive blood cultures are strongly suggestive of sinus thrombosis.

11. Transfusion of whole blood to supplement patients' resistance will save many lives.

12. Early recognition of the degree of middle ear and mastoid involvement, with rational surgical treatment, will result in a low mortality rate and the preservation of hearing.

BIBLIOGRAPHY.

1. Reik, H. O. The Blood-clot Dressing in Mastoidectomy, Considered Physiologically. Journ. American Medical Association, March 31, 1906.
2. Bárány, Prof. Robert. A New Method of Radical Operation for Chronic Ear Suppuration. Transactions of the American Academy of Ophthalmology and Oto-Laryngology, 1922, p. 62.

DISCUSSION.

DR. WILLIAM MITHOEFER, Cincinnati, Ohio: With the exception of but a few minor points, I wish to give my hearty endorsement to all of the practical points on mastoid surgery brought out in Dr. Dutrow's paper. We are undoubtedly all in agreement with the thought that the great majority of otologists of today are doing good mastoid surgery. In discussing this paper, I shall limit myself to practical points only, and not enter into any phase of the subject in which I have had no experience. It is for this reason I cannot give my approval to the Bárány method of operating, believing as I do, that when a radical operation is indicated, it should be done as completely and thoroughly as is possible.

First of all, a few words regarding mastoid pain in an individual with a chronic discharging ear. In these patients, we must not come to the immediate conclusion that the pain in and about the ear is the result of a sclerotic mastoid. Reflex causes from teeth, sphenopalatine ganglion, tonsils and hypertonic muscles of the neck must be excluded. Hypertonic muscles of the neck, with latent arthritis of the sternoclavicular and cervical joints, is a condition which is present in many patients and gives rise to a considerable amount of pain in and about the ear. If the muscles of the neck are carefully palpated during the process of our examination, a hypertonicity with painful muscles will very often be found. A patient upon whom I did a radical mastoid operation was completely relieved of earache for two months. At the end of this time, when the sternocleidomastoid muscle again became adherent to the mastoid tip, the pain returned. It was then that hypertonic condition of the muscles of the neck was discovered, and proper massage applied with complete relief. Halle reports a similar case.

I consider the skiagram as an aid in making a diagnosis to be of secondary importance. We should never lose sight of the clinical picture, which is of greater importance. I have seen patients in whom the skiagram showed little or no change, but in whom the bacteriologic examination revealed the presence of streptococcus mucosus. At the operation, much involvement of a diploic mastoid was found. So that as far as the laboratory part of the problem is concerned, I would rather draw my conclusions for operative interference from the bacteriologic, than from the X-ray examination.

I do not think it advisable to close the greater part of the mastoid wound in cases with exposed sinus or dura. This is especially true where the sinus wall is covered with granulations. I have done this many times, and until recently had no cause to regret it. During the past

year, I had a sinus thrombosis occur two weeks after the operation in a patient whose sinus wall was exposed at the time of operation, and in whom the greater part of the wound had been closed. I do not think this complication would have taken place had the open method of treating the wound been used, for the sinus wall had an apparently healthy appearance at the time of operation.

I do not agree with the essayist regarding the treatment of the tip of the mastoid. I believe that, especially in acute cases, the tip should be entirely removed, and there should remain no overhang of bone which may ultimately cause a retention pocket of pus and delay healing.

The question of recurring mastoiditis is an interesting one. It may be safely asserted that this complication is of rare occurrence when a careful and complete operation has been done. The precaution necessary to insure safety in this direction is to remove all cells, and to operate with care and less speed. Before closing the wound, it is advisable to examine carefully the field of operation and inspect again the following important regions: The zygoma, postaural region, the region anterior to the sinus wall, the tip and the posterior tip cells. If these areas are well taken care of, there is little danger of a recurring mastoiditis. In the words of Shakespeare, "Things done well and with a care exempt themselves from fear."

DR. V. A. CHAPMAN, Milwaukee, Wisconsin: Just a word about making the initial opening in the bone, particularly in patients of middle age, where the cortex is hard. I would recommend to those who do not care to use the electrically driven burr for exenteration of the mastoid, that it be used for making the initial opening in the bone. Take an electrically driven burr of about 4 mm. in diameter, and make a hole through the cortex at the selected site of entrance, and make another of the same size and in the same manner about 5 or 6 mm. below it. Then take the rongeur forcep and place one blade in one hole and the other blade in the other hole. You can then readily bite out the intervening bridge of cortex, and you have a good size opening from which to work with rongeur or curette, and no blow has been struck upon the patient's head. I think, that in mastoid cases where patients are of middle age, the use of hammer and chisel is not good surgery.

DR. HAROLD HAYS, New York City: I think none of us can disagree with Dr. Dutrow, but there are one or two points which I would like to emphasize. First, regarding blood transfusion: I think the most important thing in connection with surgery is the transfusion of whole blood. We so often have complications that we cannot quite diagnose, which are suspicious of trouble in the sinuses. Dr. Wendell Phillips, at the A. M. A. meeting this summer, cited many cases he had seen and some that I had seen, in which we were able to bring about complete recovery by transfusion. Someone made the remark, that he thought transfusions were not of much value in the acute septic infections, but the reason they were of value in the mastoid conditions was because we had had a prolonged septic condition. It is my opinion that transfusions are ideal in the type where we wish to increase the resistance of the patient.

In the mastoids, there are many cases in which we find that the

infection has closed off the attic. It seems to me it is dangerous to meddle with the attic. It is best to leave it alone. Nature will take care of it. However, we sometimes leave some infection, and in after years another attack of mastoiditis will occur; but I do not think it is right to call them recurrent mastoiditis, for if it has been properly operated, you can clear that up within twenty-four hours by a simple incision and drainage through the old mastoid wound.

In regard to the tip, I think in some cases it should be removed, but in cases where there is no great involvement, it should be allowed to remain. One point, which I have repeated very frequently since the meeting of the Triological last year, was made by Fraser, who said that no matter how careful your operative procedure is, it is absolutely impossible to remove all the necrosed bone. Some microscopic diseased bone will remain, which will take care of itself when the major part of the mastoid is carefully exenterated.

DR. L. E. WHITE, Boston, Massachusetts: I think Dr. Hays' point about removal of all the diseased tissue is well taken. We try in our work at Boston to get the cells so that they will drain, especially the posterior cell, which it is so very easy to overlook. The removal of the tip is necessary when the disease destroys its vitality. There are some cases where it is so thin that it forms a sequestrum, and in these it should be removed. If well nourished, I think it is perfectly safe to leave it. Radiographic examination helps in these cases. We have had some cases recently of what we designate as "third degree mastoiditis," that is, the cells appear practically obliterated in the plate. This usually means that the mastoid should not be opened if the discharge has practically ceased. Nature has filled that mastoid cavity with granulations, the disease is convalescent, and if you let the thing alone you will get recovery. Several of those cases have been opened, and we have regretted that we did not let Nature finish the job.

MAJOR A. EDWARD SHERMAN, U. S. Army Hospital, Hot Springs National Park, Arkansas: One suggestion recently made by Dr. Lewis Fisher of Philadelphia, in regard to an indication for operation in mastoiditis, may be of interest. He has found in a number of cases, that when he failed to get a response from the vertical semicircular canals in making the calorific test on the good or not diseased ear, the mastoid involvement had already extended to the dura. I had the pleasure of seeing one of the cases, and thought that the findings he mentioned in a paper which he recently read in Pittsburgh, and as yet not published, might be of interest to many who appreciate the value of the labyrinthine tests.

DR. ALBERT H. ANDREWS, Chicago, Illinois: In regard to opening the antrum, I wish to call attention to a point in the preservation of the hearing after these so-called simple mastoid operations. If you wish the patient to have good hearing, no matter what you do to the back part of the antrum, keep the probes and curets out of the front part. You can do very little work in the front part of the antrum without disturbing the ossicular chain, which will always result in impaired hearing.

DR. HOWARD V. DUTROW, Dayton, Ohio (closing): Since no questions have been asked, I simply wish to thank the Fellows for their generous discussion.

MANAGEMENT OF ACUTE OTITIS MEDIA AND THE PREVENTION OF THE ACUTE MASTOID OPERATION.

HAYWARD G. THOMAS, M.D.

OAKLAND, CAL.

I wish to state at the outset, to avoid misunderstanding and unnecessary discussion, that this paper refers only to the class of cases coming up in the ordinary work, and not to those in epidemics of intensity, as in the recent ones of influenza. Many of those are fulminating, and the preventive measures are usually futile.

An enormous number of cases, however, do not come under this classification, and the vast majority of these can be prevented from going to operation. The majority of mastoid cases I have operated in the last few years were those cases where the tympanic membrane was allowed to rupture spontaneously and then discharge, with little or no care until the mastoid was filled with pus—no preventive measures having been used.

In every community, north, south, east, and west, there are medical men performing mastoid operations with great frequency; indeed, all that is necessary is to have a discharging ear, and at once a mastoid operation is proposed. The argument of these is, that it is not a dangerous operation, and the quicker the mastoid is drained, the quicker the patient will recover. I cannot agree with this, and the last few years' experience has convinced me more and more that this procedure is too radical, and that if we can prevent the operation, and I am sure we can, we ought to preach the doctrine with all the power we have.

I have read papers on this subject before, and up to one year ago considered myself quite successful in preventing mastoid abscess and operation.

It was my custom, previous to that, to do a prompt myringotomy, and then give attention to the nasopharynx, etc., and get drainage. This is now a secondary procedure, since hearing Dr. Wm. L. Donohoe of Salt Lake City on the subject of avoiding myringotomy in the majority of his cases. Dr. Donohoe also read a paper in June at the meeting of the Pacific Coast Oto-Ophthalmological Society at Los Angeles, on "When is Tympanic Paracentesis Necessary?"

I prefer the term "myringotomy", which is what we mean, and not a puncture merely, which is what we do not mean.

I have adopted his method and have found that I have still further lessened the number of cases of discharging otitis media. When successful, the case is aborted at once.

Before considering these methods, if there is only a sharp earache and no bulging, I apply cold. We wish to conserve the membrana tympani whenever possible, and preventing a rupture is of prime importance. Infection comes as often from the external canal as from within.

First, cold intermittently applied. This I have found a most beneficial agent in the first stages of an otitis media, but a dangerous one if there be pronounced mastoid symptoms. Cold long applied then favors necrosis. This I find best applied by the Dr. Sprague Aural Ice Bag, filled with finely crushed ice; fifteen minutes on and fifteen minutes off. It is most grateful and in this way gives a snap to the circulation that cannot be ignored.

Dr. Donoher's method, in short, is as follows: Place a little ball of cotton, dripping with 5% cocain and adrenalin, down on the drum membrane; on top of this another, and so on till the canal is filled; then a pledget of dry cotton on top of that. This is to be repeated hourly, if necessary, till the pain has subsided. We will know soon whether our measures are going to be successful or not.

In spite of the theories of those who oppose this, that the cocain and adrenalin cannot act on the membrane, the fact remains that the pain is relieved very soon, and to my great surprise, the bulging disappears with it. This must partly be due to pressure, naturally, but cocain and adrenalin do act even on the skin when kept in contact. I have also used 10% cocain without the adrenalin, when out and having no adrenalin with me.

In the incipient cases, many have not yet had the offensive bacteria in the middle ear. The patient has blown his nose hard, or sneezed violently, and the middle ear has received a trauma—the bacteria reach it later. We are anticipating this by our preventive measures.

The rest of the measures are identical with what we do when there is a discharge or when a myringotomy is forced, so they will be taken up together.

If in a few hours the symptoms do not abate, in a small percentage of cases we are obliged to do a myringotomy. The canal is wiped out or flushed out with alcohol, then flushed out with a 2% solution of sodium citrat to prevent the blood from clotting in the wound, then a free myringotomy is done—again gently flushing out the canal with sodium citrat and sucking out the

blood and serum with a dropper; then, sometimes, I use a wick of gauze soaked in sodium citrat, but I do not use this as often as formerly, since it is as likely to act as a deterrent rather than an aid.

As often as not, infection reaches the middle ear from the external canal; I use a solution of basic fuchsin in the sodium citrat, as it is one of the most powerful bactericidal agents we have and absolutely nonirritating. As a stock solution, I keep a 1% solution—two or three drops of this to an ounce of sodium citrat are enough, as 1 in 30,000 is inhibitive to all the bacteria ever found in an otitis media.

Now, in either case, without myringotomy or with, the next measures are to the nose, throat, and mouth. The majority of our cases are in children, and otitis media is more common in children with tonsils and adenoids than in those who have had them removed. If these are present, and the evidence is to the effect that these are prime factors, I urge their removal at once and have not been disappointed.

The natural drainage from the middle ear is through the Eustachian tube, and if we have some infected and swollen adenoids in the vicinity of the mouth of this tube, it is not good surgery to allow them to remain there.

I have been obliged many times to remove tonsils and adenoids in adults when these organs were in a state of most acute inflammation, and have not yet had cause to regret the operation. The patients recover most promptly and do not have to go two or three weeks absorbing the toxins.

In cases of acute rhinitis as a cause for the ear complication, attention to the nose is of course vital.

Cleansing by gentle washing, suction applied when possible, dehydrating solutions of sodium chlorid, and sodium citrat and sometimes magnesium sulphat, are used frequently, in children using a medicine dropper.

In an adult it is sometimes expedient to mop the postnasal region with a 10% silver nitrat solution. This is too heroic for some patients, but it produces a most profuse drainage of mucus from the membranes and thus depletes them. Suction applied to the nares and the external auditory canal is of undoubted great value.

Attention to the gastrointestinal tract must not be overlooked, especially in children. Carious or dead teeth, or pyorrhea, need but to be mentioned as matters essential to be cleared up.

The next stage will be when there is a profuse otorrhea,

which is usually in those cases when the disease has been allowed to run its course without preventive measures.

As practically every case of purulent otitis media is to a greater or less extent a mastoiditis, we wish to know to what extent, and here the radiograph is of great assistance. If in our picture we find only the upper cells affected, e.g., above the canal and above the sinus, our course is fairly clear. If the cells below are involved, operation is indicated. If the opening in the membrane is not large enough, another myringotomy is indicated. Then, hot fomentations of magnesium sulphat 25% in cyanid of mercury 1/5000 are applied every two hours for forty-five minutes.

This has saved a number from mastoid operation that have been scheduled for immediate operation. In one of these cases, I opened the membrane three times in ten days to keep the discharge going—the mastoid symptoms getting less all the while, but if I had not opened the membrane, there would have been a recurrence of the symptoms.

THE PREVENTION OF SURGICAL MASTOIDITIS BY ROUTINE TREATMENT OF ACUTE OTORRHEA.

OTIS WOLFE, M.D.

MARSHALLTOWN, IOWA.

There is no surgical condition more responsive to treatment than an acute purulent otitis media, and it is my purpose to submit for your consideration a routine procedure, or system, which will not only enable the physician to dry up the discharging ear and obviate deafness, but will also prevent mastoiditis in a large majority of cases. If instituted early and carried out consistently, the mastoid operation following otitis media purulenta acuta will seldom be necessary. Even with the virulent types of streptococcus infections, such as those following scarlet fever, this routine, varied occasionally to suit individual needs, has proved highly successful. In over four hundred cases, treated during the last five years by my partner, Dr. F. I. Wahrer, and myself, the results of this routine have been uniformly the same. In a few very virulent streptococcus infections which required an early mastoid operation, we performed a simple drainage such as that advocated by Phillips¹. In the majority of these cases, the mastoid symptoms were well developed before the patients came under our care². But even if a mastoidectomy has to be performed, the routine treatment is equally valuable in conjunction with it, especially in preventing further complications.

Walter E. Brown³, stationed at Camp Green, North Carolina, during the influenza epidemic of 1918, attributes the comparatively few mastoidectomies which were necessary in their series of acute otitis medias to early myringotomy, and to the prompt abortive treatment, which was more readily carried out in the close observation of military life than in general civil practice.

It is, however, more especially to the *prevention* of conditions necessitating surgical intervention that I wish to draw your attention. Separately considered, the treatment perhaps is not new. Used collectively as a routine system, I think it presents a broader conception of acute otorrhea. For convenience of description it may be separated into three divisions: (1) General care. (2) Care of the ear. (3) Care of the nasopharynx. The same consideration is given every case early.

(1) GENERAL CARE: Every acute purulent otitis media,

whether resulting from an acute coryza or following scarlet fever or measles, should be regarded as a serious and dangerous condition. Every acute otorrhea should be considered as a potential mastoiditis. The difference is largely one of degree. There is no more excuse for neglecting pus in the head than in the abdomen. Every case of streptococcus infection should be placed in the hospital, and even mild cases confined to the house, or to bed if the temperature is at all elevated. Such complicating conditions as malnutrition, rickets, or kidney disturbance must be minutely considered, the diet regulated, and the state of the bowels carefully watched. Cod liver oil and syrup of ferrous iodid are useful in combating malnutrition and rickets, which often exist in children who appear to be well nourished. The elimination of these conditions is often of great importance in preventing an acute otitis media from becoming chronic, also in preventing the recurrence of adenoids. McCollum⁴, in discussing effect of fats on bone development, proves the superiority of cod liver oil over butter fat. From clinical observation, I am convinced of its value in otorrhea and use it as a routine in children.

(2) CARE OF EAR. The routine consists of: P (peroxid of hydrogen) I (irrigation) S (suction) A (alcohol 50% to 75% in ear and argyrol or silvol 10% to 15% in nose). I have labeled this procedure the P. I. S. A. treatment to aid in fixing it in the attending nurse's memory, as a system to be used at stated intervals for the nose and throat as well as the ear. Peroxid is instilled in the ear, and then it is irrigated with warm boric solution. Suction is next applied (we use one of the portable electric machines with glass suction tip). Alcohol is now dropped in the external canal, while the patient lies on the opposite side. Argyrol or silvol is dropped in the nose, with the patient lying on his back. If the suction is being frequently applied, peroxid need not be used every time. Irrigation is advocated instead of mopping, being safer and more easily administered by the average nurse.

Suction is the most efficient means of keeping the canal clear of pus; it also promotes hyperemia and stimulates phagocytosis (Carpenter⁵). Drainage of the middle ear is also obtained by suction. This is easily demonstrated by thoroughly cleaning the ear canal by irrigation, or mopping, then applying suction. Pus will then be noted in the canal, or exuding through the opening in the drum. It is only fair to presume that it drains the antrum as well. Phillips⁶ and Kerrison⁷ both emphasize that the antrum is the posterior end of the tympanic vault. Suction, therefore,

performs much the same function as the postauricular drainage operation of Phillips. It obtains drainage without breaking down Nature's barriers, or running the chance of infecting a new field in the mastoid. Early and free incision of the tympanic membrane is, however, absolutely necessary (Carr⁸).

When first used, suction should be rather lightly applied, the patient's complaints of discomfort or pain being the chief guide. We control the amount of suction by using a glass tip which has a hole in it over which the finger can be placed. The glass tip should be too large to penetrate the canal to any considerable depth. The patient's head should be inclined to the affected side so as to utilize the effects of gravity. Should the peroxid, or irrigating fluid, penetrate beyond the drum, it is withdrawn by the suction immediately following. Alcohol solution in the ear after suction is of secondary consideration.

In the very acute cases, we use suction every two hours; if unusually severe, as often as every hour. We use it often enough to keep the canal free from pus. In chronic cases (*otitis media purulenta chronica*) without bony necrosis, the treatment is essentially the same.

(3) CARE OF THE NASOPHARYNX is of supreme importance. Most otitis media cases have a history or evidence in the nose and throat indicative of previous trouble, such as occlusion or partial atresia of either or both nostrils, deflected septum, hypertrophied turbinates, or an old sinuitis. These nasal or pharyngeal conditions will affect the middle ear by way of the Eustachian tube, and largely influence the duration of the discharge and the tendency of the condition to become chronic. They must, therefore, be dealt with by local treatment or surgery, or perhaps a combination of both. We have found argyrol, 10 per cent, or silvol, 15 per cent, dropped in the nose every two hours, while the patient is lying down, very valuable. We have discarded all douches and sprays in its favor. It is frequently followed by phenolated petrolatum. This treatment, in conjunction with the removal of diseased tonsils and adenoids, and the routine procedure already described, often prevents the acute purulent otitis from developing. This is our first consideration in acute catarrhal otitis media (earache), together with early and free incision of the tympanic membrane if it shows signs of bulging.

Enlarged or infected tonsils and adenoid tissue are often found in adults and the same is true in sinuitis. Dean⁹ and others have called attention to its frequency in children. In children, infected adenoids and tonsils are the chief foci of infection from

which the acute purulent otitis develops. Probably 95 to 98 per cent of all cases occurring in children can be directly or indirectly attributed to diseased tonsils and adenoids, though a great deal of hypertrophied lymphoid tissue is due to such causes as malnutrition, rickets, poor ventilation, defective hygiene, etc.

In an article entitled "Does Removal of Adenoid Vegetation Prevent Disease of the Middle Ear," John Zahorsky¹⁰ takes issue with the accepted views of the otologist on this question. Though it is impossible to make a definite statement, I am, nevertheless, convinced that multitudes of middle ear diseases would be prevented, if tonsils and adenoids and their recurrences were removed at the first sign of ear involvement. It must be remembered in the treatment of either an acute or chronic otitis, that adenoids frequently recur, especially in young children. Even small pieces when they are situated on the lateral wall where they encroach on the fossa of Rosenmueller, are sufficient to be the underlying cause of an otorrhea. Adhesions from a previous adenectomy are common; they should be broken up with the finger and kept from reforming. When dealing with children, one can make a better diagnosis by feeling in the nasopharynx with the finger than by any other method. With adults, the Holmes nasopharyngoscope may be utilized.

In considering an impending or present acute purulent condition, I most emphatically recommend the radical and early removal of adenoids and tonsils, and agree most heartily with Otto Glogau¹¹, whose views are set forth in his excellent article on this subject. The extirpation of the diseased adenoid tissue alone would perhaps be sufficient in many cases, but the addition of the routine treatment is valuable and takes care of the infectious discharge, while the underlying cause is being removed.

In his practice in China, A. M. Dunlap¹² found that "following an attack of scarlet fever, no time should be lost in securing drainage . . . and thus relieve the tympanic cavity from the great strain put upon it in taking care of the discharges from the healing but unperforated mastoid." This writer, while he "does not mean to imply that every case of otitis media following scarlet fever goes on to mastoiditis" nevertheless feels that "a mastoiditis should always be suspected."

In this very troublesome and dangerous condition, where the routine treatment can be instituted early, many mastoid operations will be avoided. It treats the nose and throat infection, as well as draining the middle ear, much the same as surgery.

When the profession begins to consider and treat scarlet

fever and measles primarily as nose and throat infections with secondary skin and constitutional symptoms, we will have accomplished a big step toward reducing the vast number of complications, especially otitis media.

Harold Hays¹³, in his excellent article on "Prevention of Deafness," discusses at length the importance of removal of tonsils and adenoids in catarrhal otitis. A catarrhal otitis media, manifested chiefly by deafness, and caused by hypertrophied or infected adenoid tissue, needs only an extension of the process, or the invasion of an infecting organism, to become acute and purulent, or even to progress to a mastoiditis. The arguments advanced by Hays apply with equal force to acute purulent otitis media.

Many cases come to us with a diagnosis of acute otitis, which should more properly be classified as acute exacerbations of a chronic purulent condition, but in any case, our first thought is to consider the immediate extirpation of the diseased adenoid tissue, or if this has been previously done, to make a careful search for recurrence. If a patient has a very high temperature, with other symptoms in proportion, as in scarlet fever and measles, so that we do not feel justified in taking the chance of operation, we use the routine, intensive treatment for a time preparatory to the removal of the diseased adenoid tissue. It has been our experience, however, in some of the uncomplicated cases, where an acute mastoiditis seemed imminent, with tenderness, elevation of temperature, positive leucocytosis, etc., all indicating mastoid involvement, that immediate improvement was noted after removal of the tonsils and adenoids, or adenoids alone.

In children under ten, the only cases in which we have been required to perform a mastoid operation, where a positive indication did not exist at the time they came under our care, were those in which we did not remove the tonsils and adenoids, or recurrences, at the onset. After watching them become surgical cases requiring mastoidectomy, I am convinced that it would have been better to have taken the chance and removed the tonsils and adenoids. I do not now consider a temperature of 102° to 103° in children as a contraindication to the removal of adenoids, if there are no complications. We have had no unfavorable results, but, on the contrary, improvement without exception. In no instance have we had to perform a mastoid operation where the patient came under our hospital care within forty-eight hours after incision of the drum.

One case within three days: With this patient we did not remove the tonsils and adenoids at once.

One case, five days: Child seven, history previous earache, ruptured drum, streptococcus; admitted to hospital with temperature 105.4°; pulse 130; unconscious; meningeal symptoms. Immediate operation revealed a nonpneumatic or sclerotic type of mastoid, with very deep antrum; no fistula found. Marked improvement for two days. Succumbed to meningitis on fifth day after operation.

GENERAL CONCLUSIONS: Our experience is perhaps exceptional. Possibly it could not be maintained with a large number of scarlet fever ears, or other severe infections, as the hemolytic streptococcus, but it does seem quite significant.

We do not wish to submit this routine treatment as a substitute for the simple mastoid operation, or deprecate in the least the value of this procedure. We submit it as a logical routine to be used at the onset for the acute running ear, and emphasize its usefulness as a preventive measure in mastoiditis, and other ear complications.

When a mastoidectomy is indicated, the family physician and otologist should not defer it, but thereafter the routine P. I. S. A. treatment for the ear should be carried out, just as if no surgery had been employed. The nasopharynx should be treated in the same way, and tonsils and adenoids removed to facilitate rapid clearing up of the middle ear infection, and to abolish any chance of recurrence. Surgery will be avoided in many impending cases of mastoiditis if this treatment is thoroughly applied. I would refer you to Emerson's¹⁴ article on "*Indication for Opening the Mastoid Cortex*," which covers this ground fully.

I am going to mention very briefly a few complications we have met in conjunction with an otorrhea, and which have simulated more or less recognized indications for a mastoid operation.

1st. Young children with a persistent temperature from infected adenoids.

2nd. Children with glandular swelling and tenderness in the neck from Pfeifer's disease (glandular fever).

3rd. Periostitis of mastoid region with an increasing leucocytosis.

4th. Pain over the mastoid and in the ear from a ganglion neurosis as a complication of postnasal sinusitis as described by Sluder¹⁵. Lillie¹⁶ reports three interesting cases. I wish to mention two such with otorrhea: One classed as an acute exacerbation of chronic otorrhea, the other acute. A patient with sinusitis,

who had been treated previously for bilateral mastoid pain and earache by injection of the nasal ganglion, developed an acute otorrhea on left side, following a cold; severe pain on both sides. There was also an unerupted, impacted third molar on the right side. This case would have been very confusing if she had been a new patient.

In Conclusion: Every acute otorrhea should be accorded threefold consideration at the onset:

1st. General care. (Codliver oil in children.)

2nd. Care of the ear. Treatment of nasopharynx and ear at regular intervals.

3rd. Attention should be directed to the nose and throat first, then to the ear.

The earlier routine treatment is instituted, the smaller the chance of surgical mastoiditis.

REFERENCES.

1. Phillips, Wendell C. The Treatment of Persistent Otorrhea in Infants and Young Children by the Establishment of Postauricular Drainage. *Med. Rec.*, July 26, 1913, v. 84, p. 157.
2. Kopetsky, S. J., and Schwartz, A. A. Limitations of Reincision of Tympanic Membrane. *Arch. Ped.*, Feb., 1922, v. 39, p. 86.
3. Brown, Walter E. Ear and Sinus Complications of Influenza. *Trans. A. M. A., Sect. Laryngol., Otol., and Rhinol.*, p. 134, 1921.
4. McCollum, E. V. Pathologic Effects of Lack of Vitamin A and of Antirachitic Vitamin. Read before the joint meeting of the Sect. on Prac. of Med., the Sect. on Pharmacology and Therapeutics and the Sect. on Pathology and Physiology at the 74th An. Ses. of the A. M. A., San Francisco, June, 1923.
5. Carpenter, E. R. Suction Treatment in Acute Infections of the Ear. *Laryngoscope*, Nov., 1922, v. 32, p. 864.
6. Phillips, W. C. *Diseases of Ear, Nose and Throat.* (Text.)
7. Kerrison, Philip D. *Diseases of the Ear.* (Text.)
8. Carr, W. L. When Shall the Membrana Tympani be Incised? *Arch. Ped.*, Dec. 2, 1922, v. 39, p. 91.
9. Dean, L. W. Paranasal Sinus Disease in Infants and Young Children. *Southern Med. Jour.*, Oct., 1922, v. 15, p. 846.
10. Zahorsky, John. Does Removal of Adenoid Vegetation Prevent Acute Disease of the Middle Ear? *Laryngoscope*, Jan., 1921, v. 31, p. 22.
11. Glogau, Otto. The Chances of Cure of Mastoiditis by Tentative Tonsilloadenectomy. *Laryngoscope*, Jan., 1920, v. 30, p. 83.
12. Dunlap, A. M. Drainage of Mastoids as a Means of Preventing "Scarlet Fever Ears." *Laryngoscope*, April, 1922, v. 32, p. 272.
13. Hays, Harold. Needed Measures for the Prevention of Deafness During Early Life. *Trans. Section of Laryn., Otol. and Rhinol.*, June, 1921, p. 187.
14. Emerson, Francis P. Indication for Opening the Mastoid Cortex. *Laryngoscope*, January, 1922, v. 32, p. 278; *Boston M. and S. J.*, March 9, 1922, v. 186, p. 301.
15. Sluder, Greenfield. Headaches and Eye Disorders of Nasal Origin.
16. Lillie, Harold. Otalgia and Mastoidalgia not Indications for Operation on the Mastoid Process. Read before L. O. R. Section A. M. A., St. Louis, 1922.

DISCUSSION ON PAPERS OF DR. THOMAS AND DR. WOLFE.

DR. G. F. HARKNESS, Davenport, Iowa: I not only want to thank Dr. Thomas for having so ably presented his views on this subject, but also to thank him for having chosen this particular subject; first, because it presents an everyday problem of the otologist, and second, because it includes within its scope preventive medical and surgical practice, which is really the ultimate goal of our ambitions.

One can dismiss the well recognized prophylactic measures, the removal of diseased and hypertrophied tonsils and adenoids, as not being germane to the subject at hand.

Being confronted with an inflammation of the middle ear, the problem to my mind divides itself into two fundamentals: First, the mechanical side; second, the presence of infection. The first requires the facilitating of drainage with the least possible damage to subsequent hearing and the lessening of mastoid complications. The second considers the virulency of the infective agent as pitted against the resistance of the individual. Our problem is to correlate these so as to come to some practical conclusion for the patient's benefit.

On the mechanical side, we must remember that the middle ear, a recess in the temporal bone, closed externally by a membrane of epidermis, connective tissue and mucous membrane, and with a natural drainage through the Eustachian tube, is not a simple cavity. Besides its bony contents, the upper part is practically filled with ligaments and folds of mucous membrane.

Resolution of an inflammation in the middle ear does at times take place without rupture of the tympanic membrane. If we are to grant, which I doubt, that resolution without drainage through the tympanic membrane means the retention for a longer time of exudative products with possibly permanent damage to hearing, we must also grant that an opening through the tympanic membrane materially increases the chances of added infection to that already present.

It is surprising, in going over the literature, to find the divergence of opinion regarding the necessity for immediate myringotomy. I believe this divergence arises largely from not keeping clearly in mind the fundamentals of our problems, and failing to individualize each case.

It has been my experience, that the appearance of the tympanic membrane has not helped me a great deal in deciding whether I am confronted with an acute suppurative otitis media. With the inflammation confined to the lower half, I am inclined to consider it catarrhal, while if confined to the flaccid membrane, I consider it of the suppurative type. Myringotomy has in these cases, however, often proven it to be otherwise. I am certainly unable to definitely decide through the intact tympanic membrane when the catarrhal type merges into the suppurative type. The vast majority of cases when seen are not in their incipency, and have lost their finer points of differentiation. Therefore, I have formulated a few guiding principles.

A bulging tympanic membrane demands immediate free myringotomy, regardless of whether there is serum or pus on the other end. It is self-evident there is pressure from within the cavity of insufficient drainage

through the Eustachian tube, and if allowed to continue, means thinning of the membrane by devitalizing its tissues and inhibiting its reparative powers with the subsidence of the inflammation. Constant severe pain, with or without bulging, demands immediate myringotomy. Relief is not always secured, but certainly it is in so many cases, with so much satisfaction as to justify the procedure.

In the presence of pneumonia, diphtheria, acute tonsillitis, influenza, acute exanthemata, streptococcal throat infections, an immediate myringotomy is indicated. The individual's resistance is to be conserved to combat the general infection. The patient showing a very marked loss of hearing requires myringotomy, the loss of hearing being an indication of processes going on within the tympanum which makes the mechanics of our problem of paramount importance, demanding the most rapid and freest drainage.

Prolonged high fever, while not always indicative of the severity of the infection, does not permit of any time being lost by delaying the opening of the drum membrane.

We should bear in mind the power of certain strains of microorganisms to develop a specificity in attacking particular regions. There have been numerous instances where almost an epidemic of acute mastoiditis has occurred in certain localities. Under such circumstances, the inflamed tympanic membrane should be immediately freely incised.

The child with very large hypertrophied tonsils and nasopharynx filled with adenoids may be given the benefit of a tonsilloadenectomy, but should be watched with extreme care, and in the event rapid improvement does not take place, should have a myringotomy.

Then there is the individual with an acute otitis media who, regardless of other clinical signs, simply appeals to one's medical sixth sense as doing poorly, who should have an immediate myringotomy. Poor resistance against mild virulency is as serious as good resistance pitted against active virulence.

There are, however, a large number of cases that do not fall within the above classification, in which I believe palliative measures are justified. Time does not permit reiterating the various approved measures to be used in handling these cases. I only wish to call attention to a few, upon which at times sufficient emphasis is not placed. First, *rest*, and I do not mean only house confinement, but rest in bed. It is a common observation with an ordinary coryza, that on awakening in the morning, one often feels much better and with the upper air passages much freer. The discomfort returns as we resume and move about in our daily activities. We do not fight out a cold or infection by exercise, but we fight it out by resting and permitting Nature to marshal her resisting agents. The Army and insurance companies recognize this fact, that a man has more days fit for duty if he has bed rest during an ordinary acute otitis media. Clearer upper air passages facilitate mechanical drainage, and rest conserves body resistance.

Since the scope of the essayist's paper includes preventive medicine, we, as otologists, should continue to urge routine ear examinations in all nasopharyngeal infections regardless of symptoms, and, furthermore, rest in bed for these patients wherever it is possible.

Secondly to aid in limiting the amount of fluid within the tym-

panum, the administration of atropin to the extent of mild pharyngeal dryness is a most useful adjuvant.

I cannot see how drugs placed in the auditory canal, with its epidermal lining, are going to be of much benefit, except insofar as the vehicle used may be one that will promote osmosis through the tympanic membrane from within out. The use of carbolyzed glycerin has found rather large use, based upon the above hypothesis, the carbolic acid having, perhaps, some anesthetic effect upon terminal nerve filament in the epidermis.

The aiding of drainage by attempting nasal and postnasal medication is not always easy to accomplish with children. Temporary shrinkage of the tissues by cocain, followed by the nasal siphon (not the nasal douche) has real merit. Too much must not be expected from the various silver preparations. We have yet to find antiseptics measuring up to their test tube performances. Particular caution must be expressed against irritating medication to nasal and postnasal mucous membranes.

Inflation through the Eustachian catheter deserves more use than is accorded it. The dangers have been overemphasized. These are minimized when preceded by shrinkage of the nasal and postnasal tissues and careful siphonage, and the benefits are at times very striking.

In considering the removal of tonsils and adenoids during an acute otitis media, we must bear in mind our two fundamentals, and in so doing classify the tonsils and adenoids. First, when the otitis media is accompanied by an acute tonsillitis or adenitis; secondly, when the tonsils and adenoids are chronically infected; and, thirdly, when without apparent infection they obstruct drainage, due to their position and size. In the first, while granting that we are removing acutely inflamed foci of infection and a certain mechanical obstruction, I believe there are contraindications which outweigh the indications for this procedure.

The acute inflammation is, itself, the manifestation that Nature is marshaling her resisting forces, and attempting to form a barrier between the invading organisms and the rest of the body. Local anesthesia is unsuccessful in these cases. Under general anesthesia, there is the danger of the inhalation of infected material. The bleeding is increased materially. By breaking down Nature's leucocytic barrier, we run a certain danger of setting up severe systemic infection. Greater difficulties are offered to our operative technic. With the inflammatory reaction that at times follows, it is to be doubted that we have any assurance that we have facilitated immediate improved drainage from the middle ear.

The chronically infected, but not acutely inflamed, tonsils and adenoids of moderate size do not offer such a great mechanical obstruction—surely not as much as may follow their removal. Why stir up a chronic infection, more or less dormant, because a neighbor is fighting an acute inflammation? The indications are entirely different if it is a chronic otorrhea. As stated before, the child with very large tonsils and the nasopharynx filled with adenoids encroaching upon the fossa of Rosenmueller, that type of child who the first night after a tonsillectomy sleeps as he never slept before, that child with an acute

otitis media and without the previously mentioned indications for a myringotomy, should have his tonsils and adenoids removed at once.

Dr. Otto Glogau, New York City: About three years ago, I read a paper on "The Chances of Cure of Mastoiditis by Tentative Tonsillo-Adenectomy," before the Otological Section of the New York Academy of Medicine, and was then almost crucified. I was warned not to publish this paper, and when it was published, charges were brought against me and I had to defend myself. I stated then, that within two or three years, this matter would be taken up for discussion, and that the mastoid operation would before long become a rare occurrence, because due to appropriate prophylaxis and treatment, it would be necessary only occasionally. It is very satisfactory to me now to hear two papers on this subject. Dr. Wolfe's paper is most commendable in regard to the practical points it has brought out. If we regularly apply such a routine treatment as he describes, I think before long it will be unnecessary to perform the radical operation except in extreme cases.

I agree that we must take into consideration the three points, the constitutional, the aural and the nasopharyngeal. Since I have communicated with Dr. Wolfe, I have adopted his method of giving cod liver oil to all patients with middle ear disease. I believe there is no better remedy for the treatment of middle ear suppuration than suction. There is another method which I have used, the application of radiant heat over the mastoid. Of course, in all of these cases an early paracentesis is indicated. Drainage through the drum membrane is, however, not intended by nature. Nature's is the reverse way, drainage through the tube. Not until the secretion drains again into the nasopharynx can normal conditions be regained. We must remove the tonsils and adenoids, which are hotbeds of infection. Then we must apply suction through the nose, thereby not only removing the aural secretion, but also the secretion from the sinuses. I think the X-ray picture shows a great deal in these conditions. In every case of middle ear suppuration, I take an X-ray picture at intervals of four to five days, and thereby am able to demonstrate that we have an acute mastoiditis in just the same stages as we have in the middle ear. If we find, then, in the mastoid a swelling of the membrane or even pus, this is not as yet an indication for radical operation. If we remove the tonsils and adenoids, and in five days take another picture and find that the mastoid is clearing up, this should encourage us to continue conservative treatment. We can demonstrate through the X-ray picture improvement even to the parents. In another week, the X-ray picture may show normal conditions.

Cases of mastoiditis where meningeal or septic symptoms are present, and those showing streptococcus capsulatus as the causative germ; furthermore, those cases of mastoiditis which complicate infectious diseases, especially measles; and those cases where distinct necrosis of the bone has been demonstrated by the Roentgen picture, are excluded from the conservative procedure. In these cases, the mastoid operation has to be performed immediately. All other cases should at least be given a chance of conservative treatment, including tentative tonsilloadenectomy.

DR. E. L. JONES, Cumberland, Maryland: These are the main principles of what I have been doing for twenty-five years. For many years,

I did not dare to mention it—nearly ten years ago, I did read a paper before our Section of the American Medical Association on this method, which was rather snubbed and ignored, so it is gratifying to find these things coming to the front in a way. If I did not know from reading the various articles and visiting hospitals that there was a surgical side to these cases, I should think the whole thing should be treated medically. The main thing is not a myringotomy, but the treatment of the rhinopharynx with something sufficiently strong to set up a marked physiologic reaction, a hyperemia. That has, in my experience, a vastly more beneficial effect in controlling the inflammation than lancing the ear drum. If those cases are taken early, they run so mild a course that it amounts to nothing dangerous. The only cases which give us trouble are those which have been allowed to go neglected for a long time before we get them. If we institute Nature's method of drainage we get excellent results, and have hardly ten drops of pus from the canal throughout the whole course of the disease.

I have used cocain 10 per cent (taken from some British publication of several years ago, where it was advocated as an anesthetic for lancing the drum membrane), with adrenalin powder, 1/1000 in anilin, making a solution and putting cocain in that. In that way you get a small perforation spontaneously, which remains open as long as needed, and the invariable result is that it closes up under continued treatment, with perfect restoration of hearing, and leaving nothing but a memory for the patient. I suppose I have handled some hundreds of the acute cases and dozens of the mastoid, and have treated them in this way. If this is done, they will practically all get well. I am sure the mixture of adrenalin and anilin can be used safely, for I have never seen anything in the way of anilin poisoning but once. That was in the case of a little child, who got blue for a while, but made a good recovery. We use a mixture of 25 per cent iodine, 25 per cent phenol, 50 per cent glycerin and a little camphor-menthol. The first applications are a little bit painful, but after that they do not mind it. In many old chronic cases, where there is more or less suppuration, I have seen them get well under this treatment.

DR. HAROLD HAYS, New York City: I just wish to explain something about Dr. Glogau's paper, read before the Academy of Medicine in New York several years ago. I happened to be Chairman of the Ear Section, and took exception to the paper because he entitled it "A Cure for Mastoiditis by Removal of Tonsils and Adenoids." That is different from that given today "The Prevention of Surgical Mastoiditis." We are all agreed that there are a few conditions in the middle ear in which there is not some infection of the mastoid, and that these get well without operation. That settles Dr. Glogau's controversy, and we are just as good friends as ever.

The thing is that we have certain acute conditions of the middle ear, and these in some instances warrant paracentesis of the drum. What I wish to point out is, that anything in that way is a conservative measure to preserve hearing. I would like to ask these gentlemen who have read the papers, how often they have tested the hearing of these children following the operation? I consider that paracentesis is very important in every case. The only thing is, that we cannot keep the opening patent

as long as we wish. I think if you do this, you will find better results than with the surgical measures that have been mentioned this afternoon. I, like everyone else, resort to surgical measures when necessary, and am willing to continue that way. The statement was made, that very often an infection takes place through the ear canal after a paracentesis has been done. That may or may not be true. It has not been proved yet. I have resorted to two procedures; one, the introduction of neutral acriflavine solution before paracentesis, if possible, the keep the canal clean. In the subacute cases, whether it is necessary to do a paracentesis or not, if you apply ionization to the drum you get good results.

I did not know that Dr. Donovan had brought forth the use of cocain and adrenalin, but we have been able to avoid many operations in the middle ear by using drops of menthol gr. v, adrenalin (1-1000) 3 i, and 1% cocain up to one ounce, every hour, I think this shrinks the Eustachian tube, so that you get drainage in the normal way.

A very important point is the ability to determine the type of organism that is causing the infection, whether it is obtained from the nasal mucosa or the paracentesis opening. Whether operation will be necessary later will depend upon the virulency of the organisms and the progression of the symptoms.

As to the middle ear, I think if you use a magnifying otoscope with suction out and in, you will be able to keep the paracentesis opening patent as long as you desire without further operation. Every case is an individual one, and you have to decide the treatment upon individual merits. It depends upon your diagnostic ability whether you are going to decide right or not.

DR. L. E. WHITE, Boston, Mass.: I think one very important point brought out by Dr. Wolfe is that such patients should be taken to the hospital or kept quiet in bed. Diet is much more important than many of us appreciate, and I think patients should have everything possible done to conserve their strength. The general and local treatment to the nose and pharynx are of great importance. I think there is a time to operate in mastoiditis and a time to open the ear. The necessity of opening an ear is determined largely upon the type of infection with which you are dealing. Some infections are bound to go on to involvement of the mastoid. Those require free opening of the drum membrane. Other cases are very mild, and Nature can fight the trouble even if we do nothing. A great many forms of treatment receive credit because Nature is doing the work. I see many acute ears that subside with practically nothing done, and believe that one should have more or less of an intuitive feeling as to what should be done when he is confronted with an acute ear. I think it is much more dangerous to delay opening the drum membrane than to open it. It has never seemed to me serious to perform the myringotomy or even the mastoidectomy. We do not use irrigation for acute ears, but simply sterilize the canal, open the drum membrane freely, and then put in a strip of sterile gauze. This is changed once daily and avoids the danger of washing in infections by frequent syringing. A little absorbent cotton is placed in the external canal. The use of ice is a rather routine treatment,

ice bags being placed behind the ear. We operate on many cases and very rarely have any complications with a simple mastoid. By delaying, one many times causes serious complications. No form of treatment should be persisted in too long, as the patient's life might be sacrificed.

DR. E. H. PORTER, Tiffin, Ohio: During March and April of this year, we had in our children's hospital an epidemic of measles following closely on a very severe epidemic of influenza, which left the children in a very weakened condition. There were 140 cases of measles, and the number of cases of otorrhea as a complication was very large. Of 18 cases of threatened mastoiditis, 17 were aborted by the methods advocated by the essayists, one patient dying 6 weeks after a surgically cured mastoid from an uncontrollable epistaxis. In these cases, one of the intensely interesting things was the rapid fall of the temperature after a free myringotomy, and the difficulty we had in keeping the incision patulous and free drainage fully established. Four cases required free opening 4 times. This experience has led me to do an early free incision in the drum, repeating it when necessary, and using gentle suction in preference to syringing in this type of suppurating ears. The less one messes about in the treatment of either weakened children or adults the better, and the procedure of doing what was necessary quickly and getting out and staying out, resulted I am sure in the paucity of necessary mastoid operations in this series of cases.

DR. J. A. PRATT, Minneapolis, Minn.: I would like to make a plea for dry drainage after suppuration has been established. We make this a routine, making swabs and placing them in the canal down to the drum. These are replaced as soon as they are half filled with secretion, which means from five minutes up. You can teach the children to do this if they are old enough, and if they are not, the parents can be taught. The person to be taught watches us perform the treatment, and an explanation as how to make the swabs is given. We then supply them with a card on which is pictured the method of making the swabs, and printed instructions as to the method of applying them. The patient returns each day until we find that the swabs have been properly made and applied.

DR. NOLTON BIGELOW, Providence, R. I.: There is one point that I think deserves attention. Dr. Harkness stated that the virulence of the infection and the resistance of the patient determines whether or not a patient will recover without operation. To these two factors should be added a third; namely, the pneumatic structure of the mastoid in question. In some recent X-ray studies, carried out in collaboration with Drs. Beck and Iglauer, Dr. Gerber and I found an astonishing variation in the pneumatic development of individual mastoids. This varied all the way from an antrum deeply buried in dense bone, to the most wide spread distribution of pneumatic cells. It stands to reason that a mastoid whose pneumatic structure consists solely of an antrum deeply buried in dense bone, the so-called infantile type, will differ considerably in its reaction to infectious organisms from one that is completely pneumatic. The anatomic structure of a questionable mastoid should be determined by X-ray, for if we have this information, we are in a better position to predict the probable course of an acute mastoiditis than we are without it, while if the case does come to operation,

it is obvious that an exact knowledge of the cellular content and its distribution is a valuable asset.

DR. GEORGE MACKENZIE, Philadelphia, Pa.: I do not wish to take issue with what has been said on this subject, but it seems to me the whole thing may be narrowed down to the making of an exact diagnosis in each particular case. By that I do not mean the simple tagging of a condition, for instance, a middle ear suppuration. By diagnosis I mean one that comprehends the etiology of the particular case under consideration. This compels one to examine the patient for every thing that has been mentioned. To these I wish to add the factor of syphilis and other constitutional dyscrasias. In order to make an exact diagnosis, one must learn the exact condition of the ear, including the type of mastoid, etc. We must realize the fact that the pathologic condition in the middle ear resulted from an extension of an infection, which came by the Eustachian tube route. It is, therefore, necessary in all cases, in order to make a complete examination, to make use of a nasopharyngoscope and study the conditions about the mouth of the Eustachian tube. You will frequently find the mucous membrane red and swollen and the lumen of the tube narrowed. Upon shrinking the mucous membrane in and about the ostium of the tube, as one is accustomed to doing in the nose in the case of sinus disease, you will be rewarded now and then by seeing a drop of pus present itself at the orifice of the tube. The treatment of the Eustachian tube has been brought to your attention so often by Yankauer and a few others, that I feel that it is not necessary to repeat details at this time. Again, we must not wait for complications to arise. We must anticipate, them, and this requires most careful functional hearing tests and equally careful labyrinthine tests.

I recall that Dr. Welty of California, at the last New York meeting of the American Medical Association, said that in all cases where you consider the possibility of mastoid complications, it is necessary to make a functional examination of the vestibular labyrinth. At that time many present took exception to this. In discussing the paper I agreed entirely with Welty.

I trust that some of you have been looking for the Weber-Schwabach paradox as a sign of mastoiditis. Since first presenting the sign before this Society in Philadelphia, two years ago, I have received several encouraging reports from members of the Society, who have been conducting experiments along this same line.

DR. JOSEPH C. BECK, Chicago, Ill.: I have been much interested in the papers and discussion, but since this same subject has been assigned to me for the course of instruction, I will not try to tell you about it now, but I do wish to say that the gentlemen have been extremely cautious in their treatment, one not wishing to incise the drum, the other guarded it very carefully—and then doing a radical operation on the nose, tonsils and adenoids. I cannot see the rationale of anything of that sort. Certainly you produce a tremendous reaction and that is not good. One gentleman said "get in and get out quickly." I am sure that is a good point after the infection has subsided. We all recognize the necessity of this, but during the acute stage I certainly would

—well, you all know what I mean! I would not advise a tonsil and adenoid operation.

DR. HAYWARD G. THOMAS, Oakland, Cal. (closing): Dr. Harkness thinks there is little to be done except a myringotomy, though he agrees in the other preventative measures. Dr. Hays says in one breath that he objects to not doing a myringotomy, and in the next tells by using certain drops how to avoid this procedure. If you try my method out, you will find the hearing is just as good as if a myringotomy were done, and often it is better.

As to Dr. Beck's objections to removing tonsils and adenoids during acute inflammation; those who have not done this have no reason for objecting to it, for naturally they know nothing of it. Those of us who have done it know more about it, and have more reason for believing in it.

When I spoke of getting infection into the middle ear from the external auditory canal, I spoke particularly of those cases when the family doctor has done a paracentesis with no aseptic precautions, or when there has been a spontaneous rupture, for these cases, if left with a discharging ear, are bound to get infection from the outside.

DR. OTIS WOLFE, Marshalltown, Iowa (closing): In replying to Dr. Hays about the hearing test; I will say that I have not had the opportunity of following all these cases, but have a considerable percentage of them, enough to convince me that paracentesis does not interfere in any way with hearing. I feel that I am preserving hearing in doing an early paracentesis.

In reply to Dr. Porter; I wish to state that I do not advocate the removal of tonsils and adenoids during the active stage of scarlet fever or measles, but I do treat the nasopharynx throughout, as I consider it the source of the infection.

In reply to Dr. Beck; it has not been long since the general surgeon allowed an acute appendicitis to cause a local peritonitis before operating, for the same reasons that have been advanced against the removal of adenoids. Show me now the surgeon who hesitates to go in and get the appendix before it ruptures, because it is acutely inflamed. The surgeon now seeks to prevent peritonitis. I do not see why the same reason cannot be advanced in advocating the early removal of the source of the infection in acute otorrhea. A trial in conjunction with the rest of the routine treatment will be most convincing.

ACTINOMYCOSIS OF THE HEAD AND NECK. A REPORT OF 107 CASES.

GORDON B. NEW, M.D., F.A.C.S.

ROCHESTER, MINNESOTA.

Actinomycosis of the head and neck is probably the most commonly overlooked pathologic condition occurring in this region, as has been borne out by our experience in the Clinic. During the last 10 years, from 1913 to 1922, inclusive, 157 patients with actinomycosis have been examined at the Mayo Clinic. In 107 of these (68.1 per cent), the disease involved the head and neck; but in only 7 of this group were the patients receiving treatment for actinomycosis at the time of their examination. Some of the patients, however, had not consulted a physician. The recognition of actinomycosis of the head and neck in the Clinic has gradually increased from 2 cases in 1913 to 20 in 1922. During the last 3 years, 6 patients with primary actinomycosis of the tongue, and 2 with actinomycosis of the nasopharynx have been examined; we had not previously recognized the disease in these regions. Ninety-eight of the 107 patients were males. The age incidence was as shown in Table I.

The infection, as has been noted, is much more common in males than in females. The disease may appear at any period, from early childhood to old age. Our youngest patient was 9 years of age, and the oldest, 66. As is shown in Table I, the majority of patients were in early adult life, and almost 70 per cent of them were between the ages of 21 and 50 years. The activity of the disease bears no relation to the age of the patient.

LOCATION.

The statistics of various authors differ as to the comparative incidence of involvement of the various regions, although all agree that the cervicofacial group makes up the majority. Von Baračz, in 1903, reported 60 cases, 86 per cent of which involved the head and neck. In McKenty's series of 37 cases, 51 per cent are in the cervicofacial group. Our series of 107 cases comprises 68.1 per cent of the total of 157 cases examined at the Clinic. The primary site of the infection is shown in Figures 1 and 2, but the entire area infected is not always indicated, as often many areas were involved in the same patient. It is of interest that we have observed 6 cases of actinomycosis of the tongue in the

last 3 years, and that only 35 cases were found recorded in the literature up to 1922. A more careful study of lesions of the tongue will probably disclose a larger series.

METHOD OF INFECTION.

A consideration of the origin and mode of infection of actinomycosis in man has formed the basis for a great deal of investigation. It has been generally believed that the most common means of infection is by direct contagion from the lower animals. In taking the history of a patient suspected of having actinomycosis, we always inquire regarding the existence of a lumpy jaw in cattle in the neighborhood. The presence of this condition probably indicates that the organisms are abundant on the vegetation of the locality, rather than that diseased animals are the

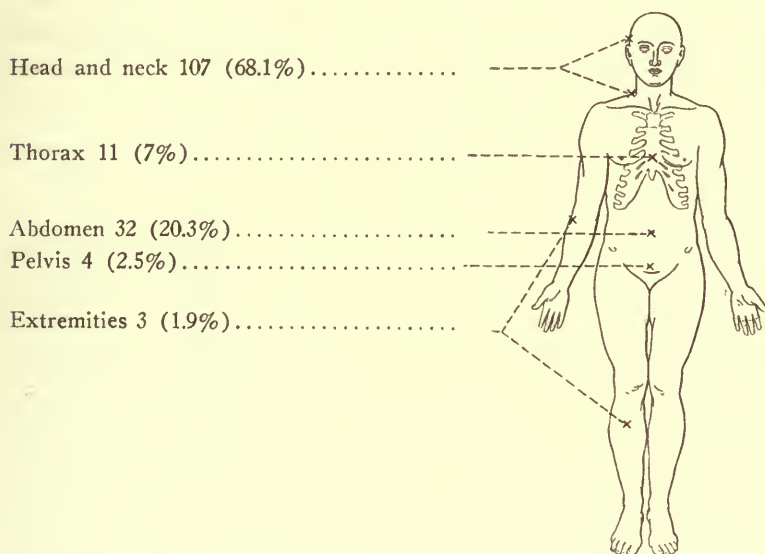


Fig. 1 Primary location of the disease in 157 cases.

primary cause of the infection in man. Transmission of the infection from the lower animals to man has been questioned. Leith denies this possibility, and it seems that this method of infection is less common, if it does exist, than was formerly believed. The 2 cases reported by Ochsner, so often cited as proof of direct infection, would seem to be little more than suggestive. Data with regard to 89 of the 107 patients in our series were obtained by means of questionnaires, and the question with regard to exposure to animals infected with actinomycosis was answered by 80. Of

this number, 45 (56.1 per cent) had not, to their knowledge, come in contact with the disease in animals. The remaining 35 patients (43.7 per cent) had been more or less closely associated with the disease in animals previous to the onset of their symptoms. In all but 7 of these, the time that had elapsed from the exposure of which they were cognizant and the onset of the symptoms varied from 6 months to 25 years, an average of 5 $\frac{1}{6}$ years. These 7 patients said that there had been lumpy jaw among their own cattle, or in the neighborhood, within 3 months of the onset of their complaint. A few of them had treated the lesions of the infected animals. Other facts, however, such as the presence of dental caries, picking decayed teeth with straws, or chewing bits of straw or grass were elicited in the history; thus, the probability of direct infection from cattle, even in this group of cases, is questionable. One of our patients with actinomycosis of the tongue had owned a cow with lumpy jaw 16 years before, but his symptoms were of only 6 weeks' duration. The nodule on his

TABLE I.—AGE INCIDENCE.

Age	Cases	Per cent
1 to 10 years	2	1.8
11 to 20 years	13	12.2
21 to 30 years	26	24.5
31 to 40 years	20	18.8
41 to 50 years	27	25.4
51 to 60 years	15	14.1
61 to 70 years	4	3.7
Total	107	

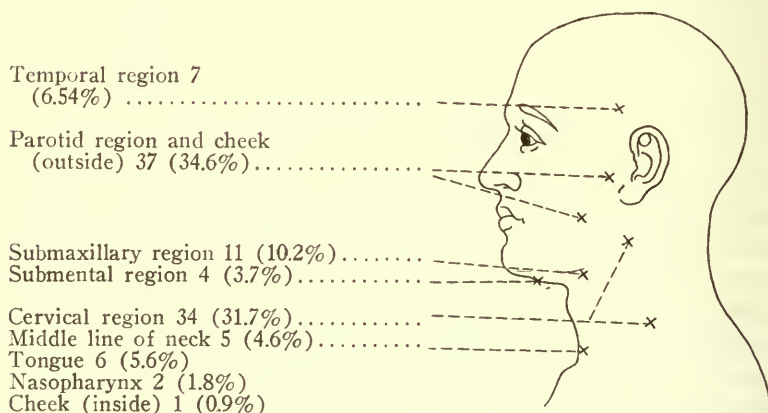


Fig. 2. Primary location of the disease in head and neck, in 107 cases.

tongue, which was about 2 centimeters in diameter, had grown to this size from 0.5 centimeter in 3 or 4 weeks. Microscopic examination following the removal of the nodule revealed a foreign body within it, apparently a splinter of wood or a grain beard, the tip of which was surrounded by actinomyces. According to Harms, this finding has been made previously in 5 cases of primary actinomycosis of the tongue in man (Schortau, Fischer, Jurinka, von Bara cz [two cases]). It has been demonstrated a number of times in other tissues of the body, namely, in the mucous membrane of the mouth, tonsils, pharynx, lungs, intestines and external auditory canal. Bostroem, by studying serial sections of actinomycotic lesions of the tongue in cattle, was able to demonstrate such foreign bodies in nearly all recent cases. In 5 of our cases, there was a definite history of a foreign body. One patient said that he had not seen an animal with the disease for 25 years, but that 2 weeks before the onset of his trouble, he had gotten a barley beard in the floor of his mouth. Pain and soreness in this region had been followed by a mass in the sub-maxillary region. One patient thought he had had a rye beard

TABLE II.—OCCUPATION.

	Patients	Per cent
Farmer	78	73.6
Laborer	9	7.5
Student	3	2.8
Clerk	2	1.8
Oil station manager	1	1.0
Meat cutter	1	1.0
Bookkeeper	1	1.0
Barber	1	1.0
Merchant	1	1.0
Stenographer	1	1.0
Tailor	1	1.0
Machinist	1	1.0
Miner	1	1.0
Cook	1	1.0
Locomotive engineer	1	1.0
Contractor	1	1.0
Chiropractor	1	1.0
Judge	1	1.0
Lumberman	1	1.0
Total	107	

in his throat, as he had a sharp, pricking sensation on one side of his throat; this was followed by a painful swelling of his neck a few days later. One patient had noticed a stinging sensation in his throat, after eating wheat, and a few days later, developed dysphagia, and a mass appeared on the side of his neck. One patient got a wheat beard in the right side of his throat, and 3 days later developed pain and swelling in the right submaxillary, cheek, and temporal regions. One patient had pain and dysphagia after injuring his tongue with a tobacco stem. In all of these cases, the disease developed subsequently in the region that had been traumatized. This would seem to indicate a direct cause of the infection. Wright believes that such a foreign body probably does not introduce the infection, but merely creates an opening, permitting organisms normally present in the mouth to gain entrance into the tissues. It would seem that, if this were true, the organisms would be found entirely enveloping the foreign body, rather than surrounding the tip of it alone, as was noticed in our case. Lord has shown that these organisms occur in carious teeth and tonsillar crypts of persons who have no demonstrable actinomycosis, so that it is probably a rather common inhabitant of the normal mouth. The infection is not infrequently seen following dental procedures. In one of our cases it developed while the patient was being treated for pyorrhea, in 6 it immediately followed the extraction of teeth, and in one, it followed the removal of tonsils. This patient had been attending an eastern girls' school. She had never been on a farm and had been in the country very little. One case of primary actinomycosis of the tongue developed immediately after biting the tongue. Four patients gave a history of an acute sore throat directly preceding the onset of their infection, although this may have been the first manifestation of the disease.

In none of our cases was there evidence of direct transmission from one person to another. McKenty and von Baračz have each reported one case which strongly suggests such transmission. Infection from milk or meat of diseased animals seems hardly probable, although one of our patients with primary actinomycosis of the tongue was a meat cutter, and Madyl reports a case of an inspector, who, while inspecting meat, frequently moistened his thumb with his tongue. He noticed a stinging pain on the area of the tongue where he had applied his thumb, and a few days later developed actinomycosis in this region. This meat, of course, was not cooked.

TABLE III.—GEOGRAPHIC DISTRIBUTION OF PATIENTS AT TIME OF INFECTION.

Minnesota	18
South Dakota	16
Canada	15
Iowa	10
Montana	8
North Dakota	7
Wisconsin	5
Missouri	5
Indiana	4
Illinois	4
Nebraska	3
Ohio	3
Washington	2
Michigan	1
Kansas	1
Oregon	1
Oklahoma	1
Georgia	1
Colorado	1
Wyoming	1
Total	107

CLINICAL HISTORY AND FINDINGS.

The clinical history of patients with actinomycosis of the head and neck depends on the virulence of the infection and the amount of secondary infection. The condition may occur as an acute phlegmon, and the symptoms not differ from this. The most common symptoms are a stiffness in the region involved, pain, and swelling. The jaw may begin to tighten and become completely ankylosed. Pain in these cases is sometimes severe and throbbing, or it may be entirely absent until the mass breaks down. In certain cases, dysphagia is an early symptom, especially if the base of the tongue or anterior cervical region is involved. In one of our cases the dyspnea was marked, because of involvement of the hypopharynx and epiglottis. Sore throat, stiffness of the neck, and earache are occasional symptoms. All types are seen, from a small recurring superficial abscess to extensive suppurating areas with multiple openings, involving almost the entire half of the head and neck. The activity of the process varies from a

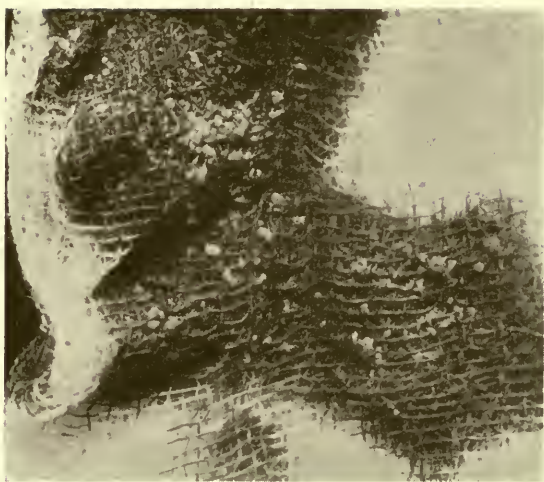


Fig. 3. (Case A346271.) Photograph of sulphur granules on a piece of gauze.



Fig. 4. (Case A325360.) Actinomycosis of the orbit, secondary to temporal region. Note the scarring in the temporal region and the exophthalmos of the right eye.

Fig. 5. (Case A203162.) Actinomycosis of the left temporal region.

slow, indolent condition, which develops in the course of months or years, to a fulminating one of a few weeks' duration. The characteristic picture of an indurated mass which later breaks down, developing multiple superficial abscesses, is probably the most common. The swelling may be a small nodule below the skin or mucous membrane, or may be a fixed, diffuse, hard mass, 12.5 or 15 centimeters in diameter. Breaking down of the involved area may occur in a few days, or the hardness may persist for months. In the cervical region, the abscesses are usually elongated, superficial and multiple, and the skin over them, which is folded in linear striations, has a soft, doughy consistency.



Fig. 6. (Case A118952.) Actinomycosis of the nasopharynx, with involvement of the central nervous system. A hard mass was present in the left nasopharynx, with bilateral choked discs; the right 2 diopters, the left 1. The sixth nerve on the right was paralyzed. The symptoms were of 6 months' duration. Two months after the first examination a sinus appeared at the left outer canthus, from which sulphur granules were obtained.

Fig. 7. (Case A359933.) Primary actinomycosis of the nasopharynx, simulating a malignant tumor. A hard mass, of 2 months' duration, was present in the left nasopharynx, bulging to the middle line. There was no external swelling. The tumor was clinically malignant. Two months later the cheek became involved secondarily, as shown in the picture.

DIAGNOSIS.

The diagnosis of actinomycosis must be based on the clinical picture, the finding of the sulphur granules, and the microscopic demonstration of the actinomyces (Fig. 3). If the disease is in the tongue, or if there is a great deal of secondary infection, a diagnosis of actinomycosis must depend on detailed study of the tissue removed for microscopic examination. In a group of cases presenting the classical symptoms and clinical picture, a diagnosis is simple. A large number of the cases, however, are



Fig. 8 (at left). (Case A411342.) Actinomycosis of the mastoid region. Note the sulphur granules in the discharge.

Fig. 9. (Case A374014.) Primary actinomycosis of the tongue, of the diffuse, hard type, involving the floor of the mouth; 2½ months' duration. Previous diagnosis of cancer, clinically.

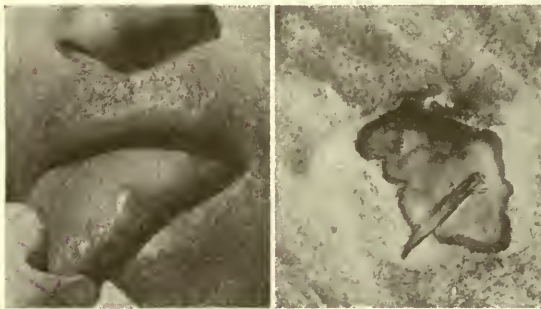


Fig. 10 (at left). (Case A416985.) Primary actinomycosis of the tongue. The nodule on the left margin of the tongue is of 1 month's duration.

Fig. 11. (Case A416985.) Low power photomicrograph showing a foreign body with actinomyces about the tip, which was found in the excised nodule from the tongue.

recognized with much more difficulty, and it is often necessary to keep the patient under observation for a time to corroborate the clinical evidence. If a fresh pocket can be opened, the sulphur bodies usually are easily demonstrated, but if there is a great deal of secondary infection, it is sometimes very difficult to obtain one for microscopic examination.

TABLE IV.—DURATION OF SYMPTOMS BEFORE EXAMINATION.

Time	Cases
1 to 2 weeks	4
2 to 4 weeks	4
1 to 2 months	29
2 to 4 months	27
4 to 6 months	10
6 to 8 months	9
8 to 10 months	5
10 to 12 months	7
1 to 2 years	4
2 to 3 years	4
Over 3 years	2
Indeterminate	2
Total	107



Fig. 12. (Case A150862.) Actinomycosis inside the right cheek, posteriorly, at the margin of the jaw, of 3 weeks' duration.

1

2

3

4



Fig. 13 (No. 1). (Case A405621.) Diffuse fulminating type of actinomycosis of the left side of the face and neck, of 3 months' duration, showing multiple sinuses.

Fig. 14 (No. 2). (Case A392156.) Actinomycosis outside of the left lower jaw. Classical location.

Fig. 15 (No. 3). (Case A367261.) Actinomycosis of the parotid region and lower jaw, a hard tumor of 1 year's duration, simulating sarcoma.

Fig. 16 (No. 4). (Case A299687.) Actinomycosis of the parotid and submaxillary regions. Note the new pockets forming in the parotid region.

In 43 cases, 46.2 per cent of our series, a microscopic diagnosis was made within 2 days after the first examination. In 66 cases, 72 per cent of our series, it was made in 1 week, and in 72 cases, 77.4 per cent of our series, it was made within 2 weeks. In 21 cases, the microscopic diagnosis was not made for a month to 6 weeks following the first examination, sometimes at a subsequent visit. Often treatment was instituted on the clinical evidence of actinomycosis, although this could not be corroborated microscopically. In 8 of our cases in which a clinical diagnosis was made, we were not able to demonstrate the condition microscopically during the patient's stay at the Clinic.

The gross appearance of the granules is quite characteristic. As a rule, they are light yellow, and about 0.5 millimeter in diameter, although we have seen them in some of our cases whitish or dark gray. It is very essential that the physician who drains the phlegmon or explores the tumor should examine carefully



Fig. 17 (at left). (Case A406422.) Actinomycosis of the cervical region simulating a branchial cyst sinus.

Fig. 18. (Case A406422.) Roentgenogram of patient in Figure 17. Bismuth which was injected into the sinus in the neck came out through the upper hole of the tonsil. The tonsil did not reveal actinomyces microscopically.

for the sulphur granules, and corroborate his diagnosis by microscopic examination. This will be much more satisfactory than simply saving pus from a drained phlegmon and referring it to the clinical pathologist for diagnosis. Sanford and Magath have recently discussed the laboratory diagnosis of this condition.

In Table V is a list of diagnoses which had been made in some of our cases, either before examination, or at the Clinic before the true diagnosis was established.

Actinomycosis may secondarily involve any part of the head and neck, and must be distinguished from many types of tumor in this region. The hard, fixed mass over the temporal region, of 3 to 6 months' duration, may be sarcoma or actinomycosis.

The subacute, subperiosteal abscess over the mastoid region and scalp may be a primary suppurative condition of the mastoid, or it may be actinomycosis. We have seen 2 patients with hard masses in the lateral wall of the nasopharynx, bulging to the middle line, with no other neoplasm of the head visible. These were clinically malignant, but both proved to be actinomycosis.

Actinomycosis of the tongue may simulate a deep seated cancer, an infected cyst, or a chronic cellulitis. We have not seen actinomycosis of the jaw bone itself, but on account of the picture presented by the involvement of the soft tissues around the bone, forming a hard mass, it must be differentiated from a periosteal sarcoma.



Fig. 19. (Case A406904.) Actinomycosis of the cervical region, simulating metastatic gland. A gland, showing clinical signs of malignancy, was removed from the anterior cervical region for diagnosis; it was reported inflammatory. Actinomyces were not looked for at that time. Later examination showed the condition to be actinomycosis. Sulphur bodies are shown in the discharge.

A phlegmon, supposedly secondary to an abscessed tooth, may be actinomycosis. In the submaxillary and cervical regions, actinomycosis must be differentiated from a chronic phlegmon, tuberculous gland, malignant lymphoma, metastatic malignant glands, phlegmon, sinus from tooth or tonsillar infections, or an infected branchial sinus. Actinomycosis in the middle line of the



Fig. 20. (Case A357702.) Bilateral cervical actinomycosis of 10 months' duration, which was diagnosed tuberculous adenitis.
 Fig. 21. (Case A354858.) Actinomycosis of both cervical regions and the back of the neck, of 1 year's duration. The diffuse hard masses were diagnosed malignant lymphoma.

neck has been diagnosed thyroglossal duct sinus, cancer of the thyroid, and tuberculous thyroiditis, in three cases (Figs. 4 to 23).

TABLE V.—DIFFERENTIAL DIAGNOSIS.

Retroorbital tumor
Malignant mass in temporal region
Subperiosteal abscess of scalp and mastoid region
Nasopharyngeal tumor, malignant
Infected cyst of tongue
Cancer of tongue
Tumor of base of tongue, malignant (?)
Sarcoma of upper jaw and parotid region
Osteomyelitis of lower jaw
Chronic phlegmon of submaxillary region, secondary to extraction of teeth
Phlegmon of cheek, secondary to pyorrhea treatment
Chronic phlegmon of cervical region, secondary to tonsillectomy
Bilateral cervical adenitis, secondary to "flu" (?); malignant(?)
Chronic indurated cellulitis of neck
Carcinoma of cervical region, recurring
Tuberculous adenitis
Bilateral malignant lymphoma of the neck
Thyroglossal duct sinus
Tuberculous thyroiditis
Cancer of thyroid

TABLE VI.—END RESULTS.

Patients	107
Patients traced	93
Not included	8
Meningeal involvement at the time of examination	6
Diagnosis only; no treatment.....	1
Death from cancer which developed on actinomycotic scar	1

Condition of 85 Patients		Patients	Per cent
Traced			
Well 5 years or more.....	9		
3 to 4 years.....	2		
2 to 3 years.....	5		
1 year	11		
Less than 1 year.....	9		
Time not stated.....	24		
	—	60	70
Dead		7	8.2
Under treatment		18	21
		—	
		85	

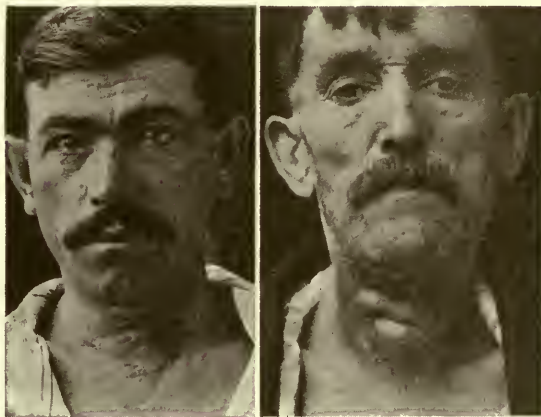


Fig. 22 (at left). (Case A360908.) Actinomycosis of the middle line of the neck, showing fixed mass in the region of the thyroid. The scars are of a previous operation for "cancer of the thyroid."

Fig. 23. (Case A409278.) Actinomycosis of the middle line of the neck, edema of the pharynx and larynx, a hard, diffuse mass extending from the submental region to the manubrium, with fluctuant areas, secondary edema of the hypopharynx and larynx, with some obstruction. Two months' duration.

With meningeal or chest involvement, additional differential problems are brought up. Moersch has reported the cases of actinomycosis of the central nervous system that have been seen at the Clinic.

TREATMENT.

The most important factor in obtaining good results in the treatment of actinomycosis is an early diagnosis. In the early cases, the patients all do well; we have not seen such patients become progressively worse during treatment. In advanced cases,

however, with extensive involvement of the head, intracranial extension may develop, or in cases of extensive supraclavicular or cervical masses, the chest may become involved in spite of treatment. Treatment is empirical. The internal administration of copper sulphat has not proved markedly effective. Arsenic was formerly used, but its good effect was apparently due to its general systemic action. The iodides are almost specific. Mercuric chlorid, phenol, tinctur of iodin, and so forth, each has been injected directly into the masses, but each has been discarded. Roentgen ray treatment is used; McKenty, however, believes that it is distinctly harmful. Recently radium has been used a great deal, and has proved very beneficial in causing a breaking down of the granulomatous masses and clearing up the induration. Surgery is a definite aid to medical treatment. The procedure we have found of most value is the opening up widely of all pockets, and packing them with iodoform gauze so that the entire area of the pocket is exposed to the air. The pockets are dressed daily, using iodoform gauze and swabbing the wound with iodin. Radium treatment is used in all cases and is repeated, as a rule, about once every 3 or 4 weeks. If the mass is hard and indurated, with no fluctuant areas or sinuses, radium is applied over the surface with 2 millimeters of lead screening, and an inch of wood distance, using from three thousand to six thousand millicurie hours in this way. This will often break down the masses and allow them to be drained. The radium also seems to block lymphatic drainage. Since we have used this treatment, none of our patients, except in the most advanced cases with masses in the supraclavicular region, has developed extension to the chest. We have also used saturated solution of potassium iodid, starting with 10 drops three times

TABLE VII.—DEATHS.

Death probably due to meningeal involvement...		2
Actinomycosis of temporal region and orbit...	1	
Death reported as due to anterior poliomyelitis and paralysis of respiratory system.....	1	
Deaths probably due to involvement of the chest		3
Huge mass in cervical and supraclavicular regions; positive roentgenograms of chest, probably actinomycosis	1	
Masses in bilateral, cervical, and supraclavicular regions, and in back of neck.....	2	
No data regarding cause of death.....		2
		<hr/> *7

*8.2 per cent.

a day, and increasing it 1 or 2 drops a dose until 200 drops three times a day are being taken. If any evidence of iodism is noticed, the iodid is discontinued for a day or two, and then resumed at the same dose. After reaching 200 drops three times a day, the patient discontinues the treatment for a few days or a week, and then starts in again at 10 drops. By this method, we have cleared up the condition in practically all of our cases except the advanced ones, in which, on account of the proximity to the skull or the chest, the infection has extended to the meninges or the thorax.

CONCLUSIONS.

The clinical picture of a rapidly growing, malignant tumor may be so closely simulated by actinomycosis, that a clinical diagnosis of such a tumor should be guarded.

A tumor or gland of the head or neck which is clinically malignant, but does not prove so microscopically, is usually actinomycotic, and further study of the tissue may demonstrate this.

The finding of a sulphur granule on exploring a tumor, draining a phlegmon, or curetting a sinus of the head or neck, frequently clears up many indeterminate diagnoses.

A reduction in the mortality of the disease depends on its early recognition and the institution of proper treatment.

BIBLIOGRAPHY.

1. Bostroem. Untersuchungen ueber die Aktinomykose des Menschen. Beitr. z. path. Anat. u. z. allg. Path., 1891, ix, pp. 1-240.
2. Cope, V. Z. A Clinical Study of Actinomycosis. Brit. J. Surg., 1915-1916, iii, pp. 55-81.
3. Harms, C. Beitrage zur Aetiologie der Zungenaktinomykose. Muenchen. med. Wchnschr., 1920, lvii, pp. 903-904.
4. Krymow, A. P. Die Aktinomykose der Zunge. Arch. f. klin. Chir., 1910, xcii, pp. 1142-1165.
5. Lord, F. T. The Etiology of Actinomycosis; The Presence of Actinomycetes in the Contents of Various Teeth and the Tonsillar Crypts of Patients Without Actinomycosis. J. Am. M. Ass., 1910, lv, p. 1261.
6. Mattson, W. W. Human Actinomycosis. Surg., Gynec. and Obst., 1922, xxxiv, pp. 482-494.
7. McKenty, F. E. A Study of Cases of Actinomycosis. Am. J. M. Sc., 1913, cxlv, pp. 835-857.
8. Moersch, F. P. Actinomycosis of the Central Nervous System. Arch. Neurol. and Psychiat., 1922, vii, p. 745.
9. New, G. B. Two Unusual Nasopharyngeal Tumors. Laryngoscope, 1922, xxxii, pp. 99-102.
10. New, G. B., and Figi, F. A. Actinomycosis of the Tongue. Am. J. M. Sc., 1922, clxii, pp. 507-517.
11. Ruhrah, J. Actinomycosis in Man, etc. Ann. Surg., 1899, xxx, pp. 417; 605; 722.
12. Sanford, A. S., and Magath, T. B. The Etiology and Laboratory Diagnosis of Actinomycosis. Minn. Med., 1922, v, pp. 71-80.
13. Baracz, R. von. Report of 60 Cases of Actinomycosis. Ann. Surg., 1903, xxxvii, pp. 336-340.

DISCUSSION.

DR. JOSEPH C. BECK, Chicago, Illinois: It is very difficult for anyone to discuss a paper of this kind. As Dr. Thomas said, if you don't know anything about it, don't discuss it. I might as well sit down, except to state that I have reported a case of actinomycosis of the middle ear. That is the only one I have seen in this region, but we have had a few cases in the head and neck region. In most cases, a diagnosis of malignancy had previously been made, sarcoma in the young and carcinoma in the older individuals.

There is just one point I wish to ask about—what is the matter with the X-ray in the treatment of this condition? Not everyone has radium, and may not the X-ray do the same thing?

DR. J. M. WAUGH, Cleveland, Ohio: A recent case which has come under my observation presented the following pathologic condition: Vincent's angina plus actinomycosis. The lesion was behind the posterior pillar on the right side. Its base appeared to be hypertrophied lymphoid tissue so often seen in this locality, but microscopic examination showed it to be an endothelioma. The patient received a deep X-ray treatment. Radium was embedded in the growth, and later local measures used to destroy the Vincent's infection. Potassium iodid was given in full dosage. He is today apparently well. Whether or not recurrence of the endothelioma will manifest itself, I do not know.

DR. THOMAS E. CARMODY, Denver, Colorado: It seems strange to report the small number of cases I have seen after listening to Dr. New. I have seen four cases, for one of which I wish to thank Dr. New, for it was one of his. Three of those cases have recovered under potassium iodid, and the fourth may be well. I have not seen her for some time, but Dr. New may have. We see very few of these cases in my locality in the human being, although there are many in cattle in that region. I have seen a large number of cattle so affected, but as I am not a veterinarian I have not treated them. (Laughter.)

DR. L. E. WHITE, Boston, Mass.: I would like to know how long the patients are under treatment, and whether it is possible to recognize the sulphur granules macroscopically. I am very glad to have heard this interesting presentation.

DR. J. A. DONOVAN, Butte, Montana: I have discussed this subject before the Cattlemen's Association from the standpoint of the physician, and will take advantage of an opportunity to discuss it before physicians.

I rather question Dr. New's statement that there is no contagion from the cattle when 73 per cent of his cases occurred in farmers.

The veterinarians inject iodine at intervals with a syringe into the growth, and give iodine salts internally, and within a short time it disappears. The question is whether the value of the cattle is great enough to justify the treatment. It has been claimed by the U. S. inspectors that the disease is purely local, and does not injure the meat, except when it becomes extensive.

DR. GORDON B. NEW, Rochester, Minnesota (closing): Regarding Dr. Donovan's point that 73 per cent of the cases of actinomycosis are in farmers, will say that a very large percentage of the total number of patients that come to the Clinic are farmers. This, of course, does

not disprove the theory that the infection comes directly from the grasses and foods to the patients, rather than that they are infected from cattle.

Regarding the diagnosis of the sulphur granules, this is usually quite a simple procedure. A little granule which looks much like a small chip of sulphur is placed on an ordinary slide and washed to get rid of the pus around it, and then covered with a cover slip without any staining, and the typical picture of the ray fungus is readily seen. While all of our diagnosis are verified by the microscope, in most of the cases a diagnosis from the sulphur granules grossly can be made.

In regard to Dr. Beck's question, I believe that X-ray would be just as good as radium in the treatment. We prefer the radium in these cases, but that is simply a matter of choice.

LARYNGECTOMY WITH RESULTS IN SEVENTEEN CASES*.

FIELDING O. LEWIS, M.D.

PHILADELPHIA, PA.

Since the title of this paper was given, four other cases have been added, making a total of seventeen laryngectomized cases performed since September, 1919.

I may, perhaps, be somewhat premature in venturing to report before this Society the results in this series of cases representing, as they do, a small experience as compared with many other surgeons, and while the period since the operation in the majority of cases is far too short to be classified as cured, yet my enthusiasm and optimism is so great for the relief afforded these unfortunate patients that I ask your indulgence.

Operable cases of carcinoma of the larynx should be treated surgically when possible. While the intrinsic laryngeal carcinomas offer by far the greater per cent of favorable results, yet all extrinsic carcinomas are not inoperable. A very large percentage are hopeless when first seen by the laryngologist. Some, however, that were formerly considered hopeless are now saved by surgery. If there is involvement of the arytenoids, upper part of the esophageal wall, and only a few of the cervical lymph nodes, with no evidence of mediastinal or lung metastasis, and the patient is a good surgical risk, I am of the opinion that surgery, in conjunction with a postoperative application of radium or deep X-ray therapy, offers a fair chance of recovery.

Of the seventeen cases operated upon, eleven are still living. The longest period since the operation is three years, an extrinsic case, and the shortest period is two weeks. There were no surgical deaths. Of the six deaths, two died of recurrence, six and eight months after operation; one died of metastases in the lungs and mediastinum four months after operation. One died very suddenly, one week after operation, from what was thought to be an embolus. One died three weeks after the operation as the result of the use of radium seeds at the time of operation. One died of a tracheal fistula two months after operation, which re-

*The technic of the operation and the cases were shown by lantern slides. Two of the cases were exhibited.

sulted from an attempt to dilate a surgical constriction of the esophagus.

The oldest case was sixty-eight years of age, and the youngest twenty-nine years of age. Three of the series were women, all of whom are living. Two of the cases, both men, have a fairly distinct speaking voice, the others a whisper easily understood by those in constant association with them. Most of the cases have resumed their former vocations, and in all but two the mental attitude is cheerful, and they seem quite happy. In two cases, it was necessary to perform eight emergency bronchoscopies for the removal of the inspissated blood and mucus which had collected at the bifurcation.

Diagnosis by biopsy was made in all the cases. Two cases healed by a primary union.

SURGICAL PROCEDURE: A complete general physical examination is most essential and oral asepsis carefully instituted. No narcotics of any kind are administered before or after operation, so as not to dull the cough reflex, which greatly aids in keeping the trachea clear of blood and secretions. A good, careful anesthetist is indispensable. I prefer general anesthesia for the entire operation. Two of the cases were operated upon under rectal anesthesia, with most gratifying results. This is a time consuming method, requiring prolonged preparation and expert care. It is difficult to find one sufficiently trained in its administration to be effective. Intratracheal anesthesia was used in most of the one stage operative cases, up to the point of dividing the larynx from the trachea, and then chloroform was substituted for ether. In the two stage operation, chloroform, when administered by an expert anesthetist, is, I believe, the most satisfactory.

Nine of the cases were operated upon by the two stage operation, and eight by the one stage. I have a preference for the one stage operation when it is possible, on account of operating in a less septic field, affording a better chance of primary union, and it affords better management of the tracheal stump.

The operation is virtually the one described by McKenty in the A. M. A. Journal of 1917. The usual T-shaped incision is made in the middle line, extending from the hyoid bone to the sternal notch, and laterally, about two inches on each side, at the hyoid bone, or upper end of the incision. Where there is metastasis of the cervical lymphatics, the incision is also extended from the lower margin of the incision as far out as necessary. The thyroid isthmus is tied and divided. Skeletonization of the

larynx. Complete hemostasis. Division of the trachea as near the cricoid cartilage as possible. Introduction of a rubber tube of sufficient size to fit snugly into the trachea for the continuation of the anesthetic and to protect the trachea from blood. Dissection of the larynx from the esophagus from below upwards as far as possible, depending upon the extent of the involvement, severing the greater wings of the thyroid, opening into the pharyngeal cavity behind the arytenoids.

The pharynx is then packed with iodoform gauze, and the larynx is completely removed from its attachment to the pharynx, including the epiglottis. The trachea is anchored to the skin by two silk sutures. The free margin of the skin incision is then sutured to the edge of the trachea by means of interrupted silk sutures. Closure of the pharyngeal cavity by two layers of interrupted catgut sutures. The introduction of a Reh-fuss feeding tube through the nose into the stomach.

Cigarette drains are placed in the dead spaces on each side of the trachea and pharynx. Partial closure of the muscles, fascia and skin over the esophagus completes the operation.

Dressings and postoperative care. A No. 6 Jackson tracheotomy tube is placed in the trachea, and moistened bichlorid dressings are laid loosely over the entire wound.

The patient is carefully watched every minute, day and night, by nurses carefully trained in the care of these cases. The tubes are kept scrupulously clean, and the dressings are changed frequently. Only water is given through the feeding tube the first twelve or eighteen hours, and then nutritious food containing the proper calories is administered every three hours, alternating with fruit juices, broths and whole milk.

The feeding tube is left in position until the pharyngeal wound has completely healed. The cigarette drains are removed on the third or fourth day and replaced by other drains if conditions warrant it.

DISCUSSION.

DR. JOSEPH C. BECK, Chicago, Illinois: You must all have been delighted with this presentation. As Dr. Fielding Lewis said, Dr. Jackson told me, and also wrote me after he had seen a case of mine following laryngectomy, that he had changed his mind and views about cancer of the larynx, and I am so glad that our biggest man has done this. It takes a big man to change his mind. If these people who have a cancer of the larynx could hear these patients talk as we have to-day, we would not have so much difficulty in getting them to submit to this operation, which will save many lives and leave the patients able to talk, even though imperfectly. If you can assure them that their families and friends will be able to understand them, it will be the biggest thing we have learned today. The technic, the indications, and so forth all have been gone over many a time. What we need now is to know the etiology. We certainly have made great progress in carcinoma, so far as treatment is concerned.

Perhaps you may be interested in our statistics in the years we have been doing this work, so I have made a little summary. I have seen in private practice 176 cases of carcinoma of the larynx, of which two were women. Of these two in which I had done a laryngectomy on Dr. Evans' basis of microscopic pathology, I found in one case the laboratory reported the Wassermann reaction on the blood three plus (+ + +), so perhaps her case was not malignant, although we know we have the combination of syphilis and malignancy. Formerly, I have been closely attached to the men who advocate laryngotomy, splitting the larynx, etc., and I am sure that if I had done laryngectomy on more cases instead of these other things, I could show a greater number of lives saved. I have twelve cases of laryngectomy that have recovered from the operation. One died of intestinal disease a year after operation, but there was no sign of carcinoma anywhere postmortem. I have four cases of laryngectomy that lived after repeated previous operations on the larynx.

If I had a cancer of the larynx, no matter how extensive it was, it would have to be operated upon by laryngectomy, pharyngectomy, gastrostomy, or anything else so that food could get down into the stomach.

The question is, where is a laryngotomy indicated? I think it is indicated, especially with the knowledge of the window resection of the larynx, if you can get a case early and have a unilateral carcinoma; a biopsy made with the consent of the patient to be operated immediately afterwards is valuable. Do not allow the patient to disappear, for the biopsy may stimulate the growth. If there is an epithelioma that is well differentiated and not mixed, and if it is a type of case where you can do an exposure of the larynx and take out a great portion of the healthy mucous membrane with the cancer, and where there is no evidence of glandular involvement or enlargement, I think the case may stay well with just a laryngotomy. Besides that, we have the radium and X-ray to follow up with. In the event of recurrence, we still can do a laryngectomy with gland resection en block.

Just a word about the radiotherapy. I had a case of carcinoma of

the larynx referred to me, in which the doctor insisted that a preoperative lethal dose of radium should be applied. He meant lethal to the carcinoma, but as a result of a twelve hour exposure before operation and six hours afterward, the patient developed a typical picture of radium toxemia. That condition simulates no other toxemia. The patient, while still in the operating room, had two attacks of propulsive vomiting and air hunger, and fifty hours after the operation he died, no doubt from the excessive dose of radium.

I am very happy that I have been here and have heard this paper of Dr. Lewis. I am sure that none of us will think that carcinoma of the larynx is so hopeless, after this presentation.

DR. GORDON B. NEW, Rochester, Minnesota: I enjoyed Dr. Lewis' paper very much, because it brought out many points of interest. I have always felt that certain malignancies about the head and neck should be classed in certain groups; some should be treated with radium, and some with radium and surgery. The operable malignancies of the larynx I believe should be surgical, if the patient's general condition will permit. It is only by showing such a group of cases as Dr. Lewis has, that the good results of surgery in this type of work can be established. While the radium is of value, it should not be used in cases that are operable, except in addition to surgery.

DR. FIELDING O. LEWIS, Philadelphia, Pa. (closing): I wish to emphasize what Dr. Beck and Dr. New have said about radium. Three or four of the cases in this series had been treated by radium before the operation, and my feeling is, that in each case the operation was made a great deal more difficult, without there being any appreciable influence on the malignancy. I believe, however, that it is often useful when used postoperatively.

NEW INSTRUMENTS AND APPLIANCES.

A TYPE OF ENUCLEATOR THAT ISOLATES THE TONSIL AND ITS CONTENTS FROM THE MOUTH AND PHARYNX DURING TONSILLECTOMY.

NOLTON BIGELOW, M.D.

PROVIDENCE, R. I.

During tonsillectomy, as usually performed, serum, blood, pus and caseous plugs are extruded from the tonsil into the mouth and pharynx. It is selfevident that this septic material should neither be swallowed nor inhaled, particularly the latter, in view of the possibility of lung abscess. Though in nowise minimizing the prophylactic value of sponging or suction, as ordinarily employed, it is, nevertheless, a fact that "the beans have been spilled" before these measures come into play. The instruments herein presented attack this problem directly in that they completely isolate the tonsil and its contents from the mouth and pharynx.

The basic principle is the same in each instance; namely, a cup attachable to a suction apparatus and a cutting member that is slidable across the mouth of the cup. As the cutting member may take the form of a flexible loop of wire, a rigid ring, or a knife, with or without a crushing blade, this principle is applicable to most of the fenestrated instruments now on the market. The word "Vacu-tome" is suggested as a generic term for instruments of this type.

In the first instrument (Fig. 1), the cup projects beyond the cannula of the snare. Its rim is elevated and beveled toward the center, while a loop of wire surrounds the base. The ends of this wire are carried through the cannula and attached to the slidable part of the snare in the usual manner. In operation, the cup is clapped over the tonsil, and fifteen to twenty pounds of negative pressure applied. As the rim of the cup is elevated, no air can leak in through the cannula of the snare, hence the tonsil is sucked into the cup without any other manipulation. It is then severed from its attachments by tightening the snare, the beveled edge allowing the loop to glide up and across the mouth of the cup. Meanwhile

serum, blood, pus and caseous plugs from the tonsil itself are sucked into a wash bottle instead of being extruded into the mouth and pharynx.

This instrument will remove the tonsil en capsule and, if sufficient care is not exercised, a portion of one or both pillars as well. This accident can be avoided with comparative ease, for by discontinuing suction just before the snare is drawn home, any pillar that may have been caught in the cup can be released instantly. Though ideal in some respects, it is doubtful, by and large, if a tonsil can be disembedded by negative pressure as safely as by digital manipulation. At all events, I do not

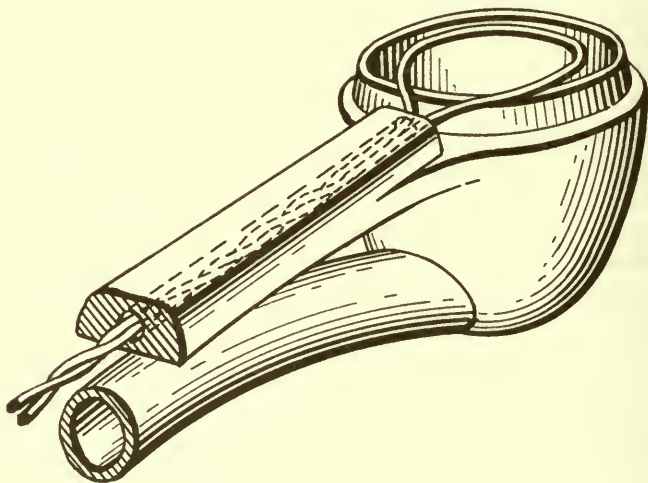


Fig. 1.

feel justified at the present writing in turning this instrument loose on any unsuspecting public, and am presenting the same merely as a matter of record.

With the Beck model (Fig. 2), no such objection arises, since no change in the Beck technic is necessary or desirable till after the tonsil has been forced through the fenestrum—which here constitutes the rim of the cup—and the snare has been drawn taut and locked. If suction is then applied, no contiguous tissues can possibly be drawn in, while at the same time all septic material from the tonsil is deposited in a wash bottle instead of the mouth or pharynx. Moreover, the tonsil itself is held more securely than by any form of tenaculum. Though desirable, suction is not an absolute necessity, providing the cup be kept rim side up.

There is always more or less difficulty in reforming and replacing the loop of the Beck snare, once it has been drawn into the cannula. This difficulty has been obviated by slotting the bottom of the ring and the sides of the cannula, the slot itself extending further down the cannula than appears in the illustration.

A working model of this instrument was submitted to Dr. Beck, who saw sufficient merit in the idea to allow me to present his instrument.

The Sluder model shown in Fig. 3, has all of the advantages above enumerated, while the application of negative pressure is

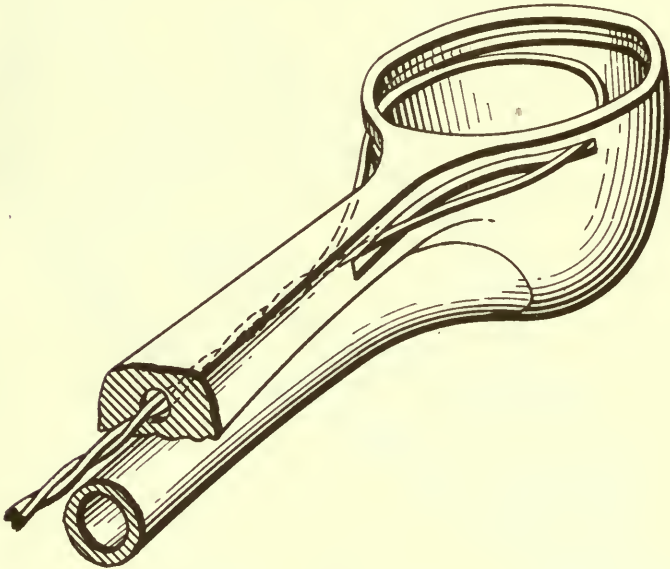


Fig. 2.

not so necessary as in the Beck model. Since the blade of the knife completely closes the mouth of the cup, the suction tube might be eliminated entirely. It seems desirable, however, in order to prevent the fluid contents of the tonsil from escaping into the pharynx. In using this instrument, there should be no change in the Sluder technic till after the tonsil has been tightly engaged by the knife. If the operator so desires, suction can then be applied while the tonsil is being severed from its attachments. After seeing two rather crude experimental models, Dr. Sluder has kindly permitted me to present his instrument.

So far as the vacuum cup is concerned, the Sauer model is to all intents and purposes, the same as the Sluder (Fig. 3). Dr.

Sauer has kindly allowed me to include his instrument in this list, after having seen a working model of the same.

It is selfevident that the vacuum principle is equally applicable to instruments having a crushing blade. To be of any use, on what is probably the most popular of these instruments, certain changes in design are necessary; for, as at present constructed, there is a gap at the proximal end of the fenestrum when the blades are driven home.

In the case of one of the newer "hemostatic" instruments, there is no apparent reason why the vacuum cup should not

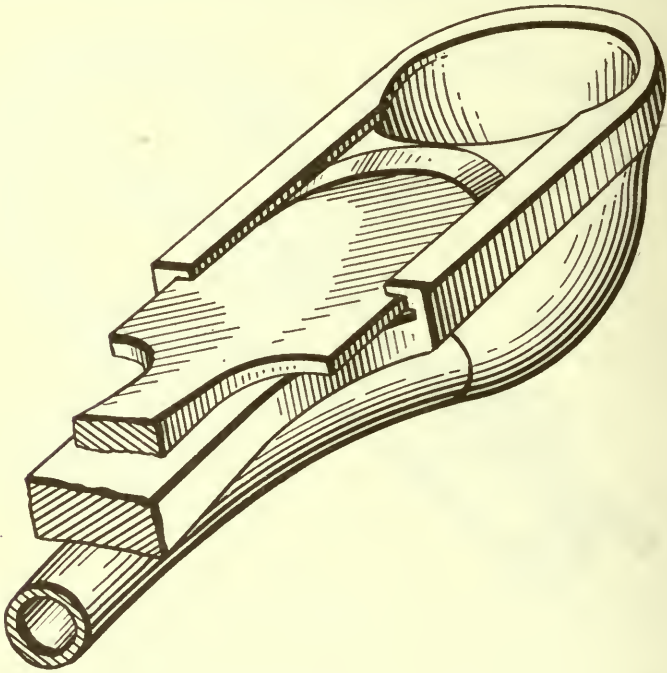


Fig. 3.

prove satisfactory. This instrument was tried out with the idea of applying a vacuum cup, but this was not done as its hemostatic properties were, in my hands, inferior to those of the new Sluder.

I have experimented with another instrument which is of different design from any of the above. So far as I could determine, the vacuum cup was entirely successful. I notified the inventor of the instrument and at his request sent him the experimental model. As he did not fully approve of the idea, I do not feel at liberty to describe the same.

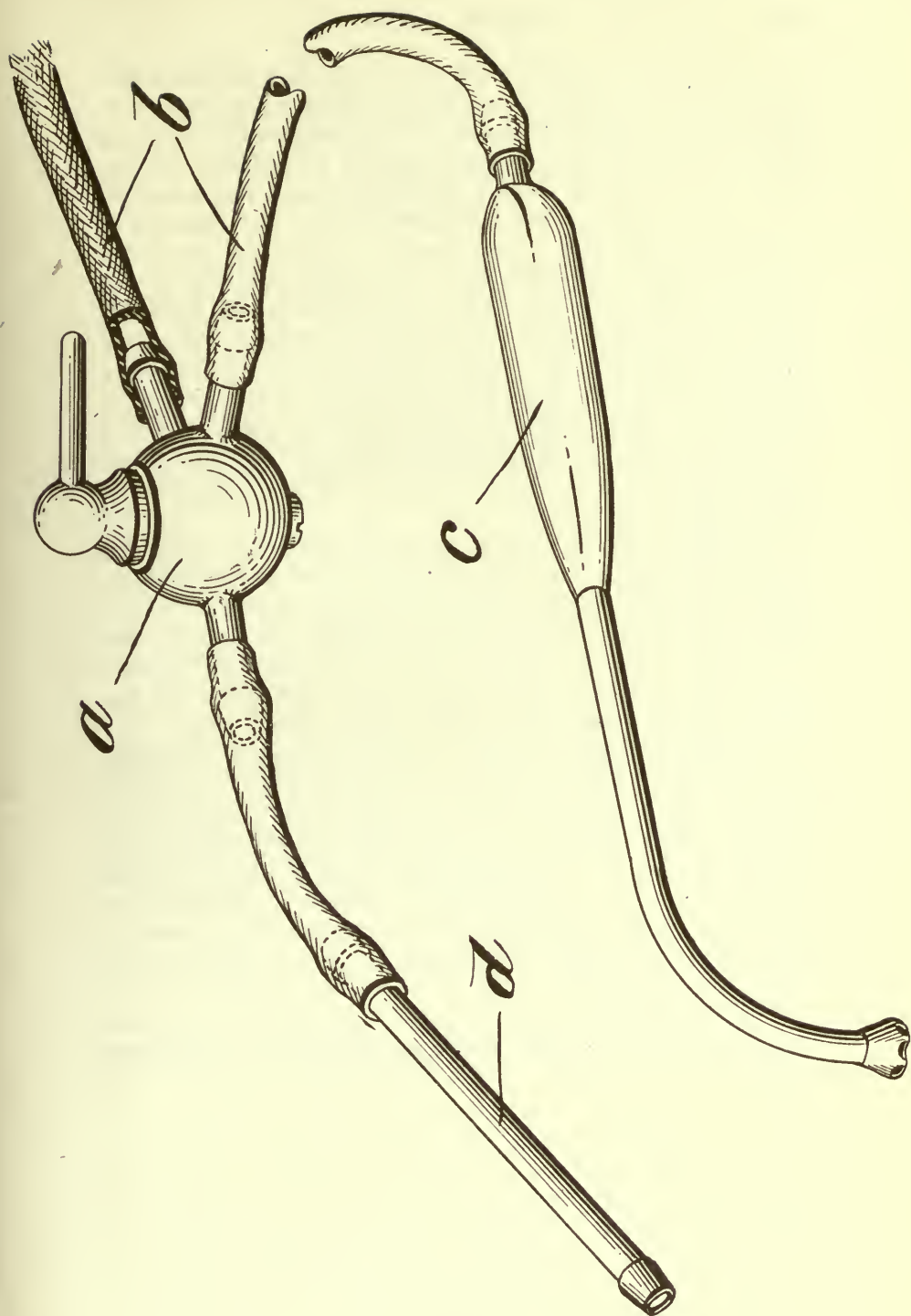


Fig 4.

As I prefer to operate without an assistant, it was necessary to devise some means for controlling the suction apparatus. The three way cock (Fig. 4a) meets this need admirably. With this device negative pressure can be applied or discontinued instantly by simply turning the valve, while both the tonsil instrument and an ordinary suction tube are available without the necessity of disconnecting the one or the other. To have the two tubes (Fig. 4b) of a different color is an added convenience in this regard. When in use, the cock can be anchored to the operator's gown or any other convenient place by two safety pins applied above and below the Y respectively. As regards suction tubes, I prefer the modified Yankauer (Fig. 4c). The metal tube (Fig. 4d) for connecting on to the unsterile hose line is more satisfactory than any form of glass connection. Flushing out the hose line with cold water will keep it from being clogged with blood clot.

Summary: As to the first model described, it is doubtful if any such instrument ever will enable a tonsil to be sucked into a cup and then severed with perfect safety to contiguous tissues. So far as the vacuum cup is concerned, the other models are entirely free from this objection, for negative pressure never is or never should be applied till after the tonsil has been completely engaged by the cutting member.

Enough experimental models have been constructed to demonstrate that the vacuum principle is applicable both to the guillotine and the ringed snare, while actual trial has shown that the cup itself in nowise alters the general characteristics of the original instrument. It is, therefore, purely a matter of choice as to which model is preferable for routine use. With any one of these models, it is possible to remove tonsils with the assurance that their septic contents will be aspirated into a wash bottle and not into the lungs.

In conclusion I wish to express my appreciation to Drs. Beck, Sluder and Sauer for their hearty cooperation in this experimental work.

WAX AND PLASTER CASTS OF THE NOSE.

JOSEPH C. BECK, M.D.

CHICAGO, ILLINOIS.

At this time, when so many rhinologists are interested in plastic work and facial deformity, I think it is well to call attention to this overenthusiasm and save some of these men some trouble. These cases are increasing in number as the publicity goes on, and we do have a great deal of trouble. In other words, individuals without any deformity whatever are the pests of the rhinologists who are doing good work in this line.

The point I wish to make is, that you must have good records, several different kinds of records. Photographs and so on we have known about, but a simple way to keep a record is to have a plaster cast made of the nose itself. Then you have a wax cast made of the nose showing the reconstruction of the nose in the way the patient wishes to have it fixed, and another of the way you think you can fix it.

If you do this, you will find that oftentimes those patients whom you do not wish to operate upon will leave you without your having made all sorts of excuses. Another point is, that when those patients see their nose as it is originally and as you reconstruct it, they decide that their nose is not so bad and drop the whole matter. However, if you do reconstruct it, and you have the reconstructed models made, they will help you a great deal later on in doing away with trouble.

TRANSACTIONS
OF THE
TWENTY-EIGHTH ANNUAL MEETING

OF THE

**American
Academy of Ophthalmology
and Oto-Laryngology**

MINUTES

REQUIESCANT IN PACE.

Dr. T. O. Edgar, Dixon, Ill.

Dr. Juan Santos Fernandez, Havana, Cuba.

Dr. C. H. Francis, Chicago, Ill.

Dr. J. W. Hadley, Frankfort, Ind.

Dr. J. R. Hoffman, Chicago, Ill.

Dr. M. B. MacLean, Chicago, Ill.

Dr. J. H. Martindale, Los Angeles, Cal.

Dr. J. W. Murphy, Cincinnati, Ohio.

Dr. W. N. Sharp, Indianapolis, Ind.

Dr. W. D. Shields, Holdredge, Nebr.

Dr. H. M. Starkey, Rockford, Ill.

Dr. A. N. Strouse, New York, N. Y.

Dr. J. Weinstein, New York, N. Y.

Dr. C. W. Wilkowske, Chippewa Falls, Wis.

Dr. C. A. Wishart, Pittsburgh, Pa.

MINUTES

October 16, 1923

The opening session of the Twenty-eighth Annual Meeting of The American Academy of Ophthalmology and Oto-Laryngology was called to order at nine-forty by the President, Dr. T. E. Carmody of Denver, Colorado.

THE PRESIDENT. We have with us this morning a gentleman who needs no introduction to this audience, a man who has worked all his life in the medical profession. Now he is working for his Government. Dr. Hubert Work, as you know, has occupied all the offices of importance in the American Medical Association; he has been Postmaster General, and now he is in President Coolidge's official Cabinet as Secretary of the Interior. I feel especially proud to introduce Doctor Work because he is a lifelong friend, and comes from my own State of Colorado. Doctor Work. (Applause)

For Address of Welcome, see page 1.

THE PRESIDENT. I am sure that we have enjoyed our welcome to the city, and we are glad to have Doctor Work with us. We know he is a very busy man, and he told me yesterday that he has the hardest job he has ever tackled. He said he thought he was busy when he was Assistant Postmaster General; he knew he was busy when he was Postmaster General; and he is quite sure he has much more to do now.

We will have a welcome now from the official representative of the doctors of Washington, Doctor W. H. Wilmer.

For Address of Welcome, see page 5.

THE PRESIDENT. We thank you, Doctor Wilmer. If Sir William Lister is in the house, will he please come to the platform? I am glad to present to you Sir William Lister of London. (Applause)

SIR WILLIAM T. LISTER. Mr. President and Gentlemen: I thank you very much, indeed, for your very kind welcome. It is a very great pleasure to me to be here, and I feel it a very great honor to be a guest. It is more blessed to give than to receive, we are told, but I fear on this occasion I have very little to give. However, I expect to learn a great deal. I thank you for your very warm welcome. (Applause)

THE PRESIDENT. Mr. Holmes Spicer is another guest from the British Isles. (Applause)

MR. HOLMES SPICER. I thank you most sincerely for your very warm reception. I feel rather abashed in addressing such a large audience. We do not have these numbers in the Old Country. I do not think the Old Country could supply anything like the wonderful audience I see before me. I have nothing to say at the present moment, but to thank you again for the extremely kind reception you have given me. (Applause)

THE PRESIDENT. I am sure we all appreciate the presence of these men at our meeting. We will now have the reading of the Minutes of the last meeting.

It was moved by DR. L. C. PETER that the reading of the minutes be dispensed with. Motion seconded and carried.

THE PRESIDENT. Our Secretary, Dr. L. C. Peter, will now make his report.

SECRETARY'S REPORT

Mr. President and Fellows: The total membership of the Academy is 1,409. During the year there were fifteen deaths. In this number is included Dr. John W. Murphy, Cincinnati, Ohio, who served as Vice President of the Academy in 1912, and as President in 1913. Dr. Murphy was for many years most active in the affairs of the Academy and was in regular attendance. The name of Dr. John Howard Martindale, Los Angeles, California, is also included. At the time of his death, Dr. Martindale was not a member of the Academy, but he was a charter member and was in regular attendance upon the meetings until ill health prevented. His name is included in this list.

Ninety-seven candidates have applied for membership. Their names were published and submitted to the membership thirty days before the meeting, according to the requirements of the Constitution and By-Laws. A considerable number filed applications too late to be acted on at this meeting. It might be well to remind the membership of this requirement of the By-Laws.

Through some oversight, the names of the Committee on Laryngic Pathology have been omitted in the program. They are Dr. L. W. Dean, Iowa City; Dr. George W. Mackenzie, Philadelphia; Dr. Ira Frank, Chicago; and Dr. Gordon B. New, Rochester.

Much to the regret of all, and to the inconvenience of many

of our Fellows, arrangements could not be made with the Trunk Line Association to permit a stop over in Chicago in order to attend the Clinical Congress of the American College of Surgeons. This privilege may be obtained for future meetings by coordinated and early efforts on the part of the organizations concerned.

The Secretary would remind the members that all railroad certificates should be deposited at the Registration Bureau for validation. Two hundred and fifty certificates must be deposited before any can be validated.

Respectfully submitted,

LUTHER C. PETER,
SECRETARY.

It was moved by DR. EMIL MAYER that the report be accepted. Motion seconded and carried.

REPORT OF TREASURER

Balance on hand.....	\$ 7,193.89	
Bonds	33,578.50	
	<hr/>	
	\$40,772.39	
Received from dues, application fees, in-		
terest on bonds, etc.....	15,329.70	\$56,102.09
	<hr/>	
Disbursements	6,609.29	
	<hr/>	
Balance	\$49,492.80	
Cash	\$15,914.30	
Bonds	33,578.50	
	<hr/>	

SECORD H. LARGE, TREASURER.

It was moved by DR. ROBERT S. LAMB that the report be accepted and referred to the Auditing Committee. Motion seconded and carried.

Under the head of Special Committees, the Program Committee presented the program now in the hands of the members.

In the absence of Dr. Lucien Howe, DR. EMIL MAYER read the report of the Committee on National Medical Research Laboratory.

REPORT OF THE COMMITTEE ON A NATIONAL BUREAU OF MEDICAL
RESEARCH

In a previous report, attention was called to the improved opportunities which might be available for investigation if we had in the President's Cabinet one member charged especially with the care of public health. It would be the duty of one of the numerous bureaus of this department to coordinate as far as possible the work done in the various laboratories which now exist in different departments of the government, or in the laboratories of different institutions throughout the country.

But as any such change in the machinery of government would necessarily require discussion of its political results, and as one of the first requisites of scientific research is the preparation of bibliographies, therefore our committee showed also the desirability, meanwhile, of encouraging collections of current literature, more elaborate than that which is given by the *Index Medicus*, or by any similar publication in English.

With that in view, this Academy last year expressed its approval of any plan which might prove practicable to establish an International Year Book of Ophthalmology, with the hope of having later substantially similar year books of Otology, Otolaryngology and other departments of Medicine and Surgery.

During the past year, the Bureau of Legal Medicine and Legislation of the American Medical Association has given considerable attention to the more definite governmental control of public health. That would contemplate indirectly a National Bureau of Medical Research, including ophthalmology and otolaryngology with other branches of medicine and surgery. As the question is at present in such able hands, it does not seem appropriate or politic for this committee to make itself officious, perhaps to the detriment of the very object desired. Rather is it becoming in us simply to ask that our committee be continued in order to assist if any cooperation is asked for, or to report again if any further action by the Academy seems advisable.

LUCIEN HOWE

EMIL MAYER

EDWARD B. HECKEL.

It was moved by DR. MAYER that the report be adopted and the committee continued. Motion seconded and carried.

REPORT OF COMMITTEE ON PUBLICITY AND SERVICE

I think the committee is misnamed, because we have had referred to us all kinds of requests for conducting newspaper advertising campaigns and things of that kind. But we conceived that what we were appointed for was to approach other national societies, for the purpose of forming a combined national committee for the consideration of various subjects of general interest to ophthalmologists and oto-laryngologists. The matter was therefore put under way to be brought before each of the national societies at their annual meetings during the past year, with the following result: The Section on Ophthalmology of the American Medical Association acted favorably on this and will appoint a delegate. They have not appointed him yet, according to my last advices. The gentleman to whom was assigned the task of bringing the matter before the Section on Laryngology of the American Medical Association says that he wrote me a letter saying he could not attend the meeting, but I did not get the letter, and therefore no arrangement was made and the matter was not brought before that Section. The Triological acted favorably on the proposition and will appoint a delegate. The American Ophthalmological Association acted favorably and appointed a delegate. In the American Otological Society there was another misunderstanding, and the matter was not brought up. The American Laryngological Society considered the matter, but did not take any action.

We would like to ask that the committee be continued, and we will try to bring the matter before all the societies before the next meeting.

E. C. ELLETT, CHAIRMAN.

It was moved by DR. A. A. HAYDEN that the report be accepted and the committee continued. Motion seconded and carried.

REPORT OF SECTION ON INSTRUCTION

The report of this committee will start Thursday night, and the program for the course is already in your hands. It has been the effort of the committee to follow out the wishes of the Academy as expressed by letters and verbally by various members of the Council as far as possible, and we will try to present a program for your instruction and delight that will carry out

the underlying idea for which the Committee on Instruction was formed. There are no specific points to be taken up in this report, but I do wish to make one announcement. There has been some little confusion as to where the course of instruction will be given. It will be at the District of Columbia Medical Society building, 1718 M St.

As far as the upkeep of the Section is concerned, there has been an endeavor to make it pay its own way—not to pile up any surplus, and yet have no deficit. So far we have been successful in that endeavor.

HARRY S. GRADLE, CHAIRMAN.

It was moved by DR. WALTER LANCASTER that the report be accepted. Motion seconded and carried.

REPORT OF SECTION ON PATHOLOGY

The Committee on Pathology has been working in rather a loose manner, and unfortunately for some, the greatest endeavor has been along the line of ophthalmic pathology, so that otolaryngology has been treated in rather a stepmotherly fashion. However, it is hoped that will improve.

The Section on Pathology really should be divided into two separate committees—ophthalmic pathology and oto-laryngic pathology. The two are so radically different that no one committee will be able to handle them properly. So far, the section on ophthalmic pathology has endeavored to become nationalized, in that the Academy has joined with the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association in endeavoring to establish a National Museum of Ophthalmic Pathology, with headquarters at the Army Medical Museum. The method of procedure is rather simple. We are requesting the ophthalmologists to send in all of their specimens, not only those that are rare or unusual, for in order to have a complete collection everything is necessary. The material may be sent to the Army Medical Museum, where it is worked up by the technicians and pathologists, and a final report and diagnosis is given the donor. At the request of the donor, photographs or gross specimens as well as slides will be furnished. In this way we are gradually building up a Museum of Pathology where every specimen is available to every member, and we hope eventually it will be available to every reputable physician, both in Washington and outside, as the material can

be sent by mail. Thanks to the efforts of one of our leading members, the extremely valuable collection of Dr. Ball of St. Louis has been added to the Museum. This consists of specimens, plates, photographs, and some types of old instruments. It is the duty of all members some time during this meeting to visit the Museum and see what we are endeavoring to do.

HARRY S. GRADLE, CHAIRMAN.

It was moved by DR. A. A. HAYDEN that the report be accepted and the committee continued. Motion seconded and carried.

REPORT OF AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS

I did not know that I was to report for this Board, and have nothing in preparation except to say that the work of the Board has gone on as usual. At the last meeting in Baltimore, yesterday, we examined ten candidates, and the members of the Board were very agreeably surprised and encouraged to see the much better effort the candidates made in their examinations. I think it is undeniable that the American Board for Ophthalmic Examinations is uplifting the science of ophthalmology. It is stimulating the younger men to do better work, and it hopes that much good will come from this. When a man is conditioned in one or two subjects, he is allowed to take a special course in these subjects and then present himself to a specified examiner for further examination, so that when a man receives a certificate from the American Board of Ophthalmic Examination he is supposed to be thoroughly grounded in all branches of ophthalmology, and since a number of societies demand that a man shall have that certificate before becoming a member, the men are stimulated to come forward for examination, and to prepare themselves for examination by better work. It seems to me this Board deserves a good deal of credit from this Academy for its efforts in uplifting Ophthalmology.

ALLEN GREENWOOD.

It was moved by DR. EMIL MAYER that the report be accepted. Motion seconded and carried.

REPORT OF COMMITTEE ON EXAMINATIONS IN OTO-LARYNGOLOGY

The Oto-Laryngic Board has not been in existence as long as the Ophthalmic Board, but we are endeavoring to come up to the high standard that they have set, and this year we had thirty candidates. We spent yesterday in examining them, and the report will be sent in to the Council.

We feel, with Doctor Greenwood, that this Board is doing something for Oto-Laryngology, and that we are really selecting men who not only have reasonably good preparation for the work, but our work is increasing the number of men coming before the Board each year. We hope that in time the Board on Oto-Laryngology may have the same standing throughout the country as the Board on Ophthalmology has established for itself.

JOHN M. INGERSOLL.

It was moved by DR. EMIL MAYER that the report be accepted. Motion seconded and carried.

REPORT OF NECROLOGY COMMITTEE

The following deaths occurred during the year:

Dr. Thomas O. Edgar.....	Dixon, Illinois
Dr. Juan Santos Fernandez.....	Havana, Cuba
Dr. C. H. Francis.....	Chicago, Illinois
Dr. James W. Hadley.....	Frankfort, Indiana
Dr. John R. Hoffman.....	Chicago, Illinois
Dr. Malcolm B. MacLean.....	Chicago, Illinois
Dr. John Howard Martindale.....	Los Angeles, California
Dr. John W. Murphy.....	Cincinnati, Ohio
Dr. Walter Nevin Sharp.....	Indianapolis, Indiana
Dr. William D. Shields.....	Holdredge, Nebraska
Dr. H. M. Starkey.....	Rockford, Illinois
Dr. Alfred Nathan Strouse.....	New York City
Dr. Joseph Weinstein.....	New York City
Dr. Conrad W. Wilkowske.....	Chippewa Falls, Wisconsin
Dr. Charles A. Wishart.....	Pittsburgh, Pennsylvania

The members stood with bowed heads for one minute in memory of the departed.

DR. WALTER B. LANCASTER. Under the head of New Business I would like to mention a matter.

The American College of Surgeons has appointed a committee to draw up a standard nomenclature of operations. The members of this Academy on that committee feel that it is a sufficiently important subject to be officially backed by the Academy, and moreover that it will be desirable that other associations cooperate. We, therefore, propose that the President of this society appoint two members to invite the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association to appoint members who shall

cooperate to draw up this standard nomenclature. The letter from the College of Surgeons says: "At the present time there are several complete nomenclatures of this kind, each differing from the other, and the need is to have one nomenclature that will eventually be adopted in all hospitals."

I move that the President-elect appoint a committee of two to invite the other societies to cooperate.

Motion seconded by DR. JOHN E. WEEKS, and carried.

Meeting adjourned.

October 17, 1923

The business session was called to order at nine-fifty by the President, Dr. T. E. Carmody of Denver, who announced the following members of the Auditing Committee, this committee to report at the Thursday morning business session.

Dr. James M. Patton.....Omaha

Dr. Walter R. Parker.....Detroit

Dr. C. F. Heard.....Erie

The President then called for the Report of the Council, which was presented as follows by the Senior member, DR. EMIL MAYER.

President, Walter B. Lancaster.....Boston

1st Vice President, W. P. Wherry.....Omaha

2nd Vice President, S. Hanford McKee.....Montreal

3rd Vice President, H. L. Pollock.....Chicago

Treasurer, Secord H. Large.....Cleveland

Secretary, Luther C. Peter.....Philadelphia

Editor Transactions, Clarence Loeb.....Chicago

COUNCILLORS

Frank L. DennisColorado Springs

Arthur J. BedellAlbany

NEW MEMBER SECTION ON INSTRUCTION

F. R. SpencerBoulder, Col. (4 years)

Meeting adjourned.

October 18, 1923

The session was called to order at nine thirty-five by the President, Dr. T. E. Carmody of Denver.

THE PRESIDENT. The Secretary wishes to make a report for an International Congress.

DR. LUTHER C. PETER. This report is that of the Treasurer,

and is the same report which has been made before the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association, so that the members who contributed may know the status of the treasury at the present time.

There are a number of copies of the Transactions in the possession of the Committee on Publication. These Transactions will be given to libraries upon application. The Committee has distributed them as far as possible to the libraries throughout the United States. If any have been omitted a limited number can be supplied. If there are any ophthalmologists who desire copies, they can be obtained by the payment of \$5.00 per volume.

There will be a small sum left after all expenses have been paid, and this sum will be disposed of in some proper way. For example, it might be given to the Knapp Memorial Fund or some similar fund, and this will be determined by the Committee before they finally disband.

THE REPORT OF THE TREASURER OF AN INTERNATIONAL CONGRESS
OF OPHTHALMOLOGY WHICH MET IN WASHINGTON, D. C.,
APRIL, 1922

Receipts

Contributions from members of the General Committee	\$ 1,215.15
Membership subscriptions	10,901.22
Interest on bank deposits and other incidentals.....	294.86
Sale of Transactions.....	110.00
Contributions from members to defray deficit.....	2,511.91
Total Receipts	<u>\$15,033.14</u>

Disbursements.

Printing (Preseasonal volumes, programs, transactions, stationery etc	\$10,649.72
Secretarial expenses of committees.....	450.00
Postage, telegrams and other incidental expenses.....	715.39
Local Entertainment Committee in Washington, (expenses of guests, reporters, etc.).....	3,136.61
Total Disbursements.....	<u>\$14,951.72</u>
Cash Balance, October 10, 1923.....	\$ 81.42

Respectfully submitted,

L. C. PETER, TREASURER.

REPORT OF AUDITING COMMITTEE

Your committee had examined the books of the Treasurer and find them to be accurate, with all the vouchers attached for the expenditure of funds.

JAMES M. PATTON,
For the Committee.

It was moved by DR. E. C. ELLETT that the report be accepted and the committee discharged. Motion seconded and carried.

REPORT OF SENIOR MEMBER OF COUNCIL

The Council begs leave to make the following recommendations.

Place of Meeting: Montreal, Canada, last week in September; exact date will be sent out with notice.

We nominate for the American Board for Ophthalmic Examinations, Dr. Phinzy Calhoun, Atlanta, Georgia.

We also wish to present the following resolutions.

First: Each candidate for examination in oto-laryngology shall pay a fee of \$10.00, such sum to be used to defray the expenses incident to holding these examinations.

Second: We would like to call attention to a communication received from Doctor Stucky, the first paragraph of which reads as follows:

"May I urge that you use your influence with the Surgeon General of the Public Health Service to accept the proposition of the Rockefeller Institute, through its representatives, Drs. Flexner and Noguchi, to devote special time and effort towards ascertaining the etiology of trachoma?"

Acting on this letter, the Council makes the following recommendation to the American Academy of Ophthalmology and Oto-Laryngology: We wish to call attention to the fact that trachoma is still widely prevalent and that its etiology is unknown. Investigation to determine its cause would be of inestimable value to mankind. We therefore would heartily endorse any action which the Surgeon General of the Public Health Service may undertake toward the solution of this very important problem.

WALTER R. PARKER.

It was moved by DR. HUNTER H. MCGUIRE that the first reso-

lution as well as the preceding recommendations of the Council be adopted. Motion seconded and carried.

It was moved by DR. A. A. HAYDEN that the second resolution be adopted. Motion seconded and carried.

The SECRETARY read the nominations presented by the Council at the Wednesday morning business session.

President, Walter B. Lancaster, Boston.

1st Vice President, W. P. Wherry, Omaha.

2nd Vice President, S. Hanford McKee, Montreal.

3rd Vice President, H. L. Pollock, Chicago.

Treasurer, Second H. Large, Cleveland.

Secretary, Luther C. Peter, Philadelphia.

Editor Transactions, Clarence Loeb, Chicago.

Councillors, Frank L. Dennis, Colorado Springs; Arthur J. Bedell, Albany.

New Member, Section on Instruction, F. R. Spencer, Boulder, Colorado; (4 years).

It was moved by DR. HAL FOSTER of Kansas City that the recommendation of Council be accepted, and that the Secretary cast the ballot of the Association for the names read. Motion seconded and carried. The newly elected President was escorted to the platform by Drs. Foster and Francis.

DR. WALTER B. LANCASTER: I would like to express my deep appreciation of the very great honor you have conferred upon me. A sense of my own deficiency would certainly overwhelm me were it not that I realize that important and conspicuous as the President is, he is by no means the whole works. With the Council, wise, astute and experienced, with the splendid and efficient Committee on Instruction, and above all, with our Secretary, fertile in plans and indomitable in carrying them out, patient in difficulty, and always lovable, I am not downhearted. I feel we can go forward to a year of unequaled prosperity, and I pledge you that I shall serve to the best of my ability. I thank you. (Applause).

THE SECRETARY: The Council recommends the following men to the Academy for election to membership:

Alexander, Dr. George James.....	Philadelphia
Annon, Dr. Walter Thomas	Philadelphia
Berrisford, Dr. Paul D.....	St. Paul, Minn.
Blassingame, Dr. Charles Decatur.....	Memphis, Tenn.
Bozer, Dr. Herrmann Eugene.....	Buffalo
Bruner, Dr. Abram Breneman.....	Cleveland

Clerf, Dr. Louis Henry.....	Philadelphia
Copps, Dr. Lyman Alden	Marshfield, Wis.
Curtin, Dr. Eugene A.....	Scranton, Pa.
Davis, Dr. Thomas W.....	Winston-Salem, N. C.
Dean, Dr. Alfred	Grand Rapids, Mich.
Dorge, Dr. Richard Irving	Minneapolis
Forgrave, Dr. L. Robert	St. Joseph, Mo.
Gill, Dr. G. Bache	Washington
Goldman, Dr. Harry G.....	New York City
Greene, Dr. Joseph Berry.....	Asheville, N. C.
Harris, Dr. W. Robin.....	Memphis, Tenn.
Hetrick, Dr. Llewellyn Evans.....	New York City
Holdsworth, Dr. Frank.....	Traverse City, Mich.
Howard, Dr. Lloyd G.....	Council Bluffs, Ia.
Howard, Dr. William Henry.....	St. Paul, Minn.
Johnson, Dr. Thomas Holland.....	New York City
Lerner, Dr. Macy Levi.....	Rochester, N. Y.
Lombardo, Dr. Melchoire	Brooklyn, N. Y.
Looper, Dr. Edward Anderson.....	Baltimore
Lyman, Dr. Harry W.....	St. Louis
McClelland, Dr. Carl C.....	Detroit
McGinnis, Dr. Edwin.....	Chicago
McKinney, Dr. Alexander R.....	Saginaw, Mich.
Marshall, Dr. George Guerin.....	Rutland, Vermont
Morton, Dr. Howard McIlvain.....	Minneapolis
Myerson, Dr. Mervin Carueth	Brooklyn, N. Y.
Nelson, Dr. Lloyd L.....	Norfolk, Neb.
Parrish, Major Robert Eunice.....	Washington
Peirce, Dr. Howard Wilber.....	Detroit
Proetz, Dr. Arthur Walter.....	St. Louis
Rowland, Dr. William Denton.....	Boston, Mass.
Rush, Dr. Calvin C.....	Johnstown, Pa.
Somberg, Dr. Joseph Sheldon.....	New York City
Sullivan, Dr. John Joseph.....	Scranton, Pa.
Tomassene, Dr. Raymond A.....	Wheeling, W. Va.
Townsend, Dr. John Ferrars.....	Charleston, S. C.
Tucker, Dr. Gabriel Frederick.....	Philadelphia
Walsh, Dr. Joseph Mark.....	Rapid City, S. D.
Watson, Dr. Roy Seymour.....	Saginaw, Mich.
Weeks, Dr. Webb William.....	New York City
Wendel, Dr. Jacob S.....	Detroit
Wheeler, Dr. Merritt W.....	St. Paul, Minn.
Winter, Dr. John Arthur.....	Duluth, Minn.

It was moved by DR. R. H. T. MANN that these men recommended by Council be elected to membership. Motion seconded and carried.

DR. LEE M. FRANCIS: I have the honor to present for honorary membership our distinguished guest, Sir William T. Lister of London, England.

Unanimously elected by rising vote.

THE PRESIDENT: Sir William Lister, your modesty and your great learning has taken us by storm, and we are glad to welcome you to honorary membership in the Academy. We hope you will be able to make a speech this morning.

SIR WILLIAM T. LISTER: I fear my ability to make a speech is no better in the morning than at night, but I want to thank you most warmly for the great honor you have conferred upon me by electing me to honorary membership in this splendid, live, enthusiastic association.

DR. WALTER B. LANCASTER: I move that a vote of thanks be extended to the local committee for their very effective and successful preparations for the meeting. Unanimously carried by rising vote.

REPORTS OF SPECIAL COMMITTEES

REPORT OF COMMITTEE ON STANDARDIZATION OF TUNING FORKS

To the Members of the American Academy of Ophthalmology and Oto-Laryngology: Pursuant to a resolution passed by the section on Oto-Laryngology of the American Academy at the meeting in Philadelphia in October, 1921, a committee on the standardization of tuning forks and hearing tests was appointed. This committee made a report at the last meeting of the Academy, held in Minneapolis, September, 1922.

By the courtesy of Dr. Edward B. Dench, Chairman of the Committee for the Standardization of Tuning Forks of the American Otological Society, we were again able to obtain the report of the committee of that Society. Their committee last year adopted as a "low limit the Dench tuning fork of 26 to 64 double vibrations, and a fork of 256 double vibrations for bone conduction, the latter fork being made with a broad base so as to be applied more easily to the mastoid." That committee further stated that an "investigation was being undertaken regarding the standardization of the Galton whistle, but it had been found impossible to find a maker for the stand-

ard Galton whistle except the Edelmann, made in Munich, which is very expensive." The committee then considered the possibility of having only a part of the scale standardized, namely: 6, 12, 18 and 24,000 double vibrations, so as to determine the upper tone limit, but the manufacturers who were approached did not consider this plan feasible as yet. That committee stated that the Galton whistles that are on the market at present are not accurate, but an approximate estimate may be made recording the upper tone limit in cases of slightly reduced, moderately reduced, much reduced and greatly reduced, corresponding to 24,000, 18, 12 and 6 double vibrations. This was tried at the Otological Institute in connection with the Dench fork of 256 double vibrations and acted fairly well. A blank for taking aural histories had been compiled which was a combination of the various hospital blanks, namely, that of St Luke's Hospital, the New York Eye and Ear Infirmary and the Neurological Institute.

A resume of our report made last year is with very slight changes as follows: Recommendations first with regard to the character of the fork employed. This should be well balanced, of rustless metal if such can be obtained, of a definite pitch, definite duration of vibrations, moderate in price and so made as to eliminate loud overtones. To avoid rattling of the weights, one manufacturer uses round, flat, clasp like weights in place of the projecting, easily broken thumb screws. We understand, however, that this mechanism is patented and therefore could not be used by other manufacturers without the payment of royalty.

Second: The forks should be held uniformly near the ear, preferably with the wide surface of the prongs parallel to the ear, in order to get a maximum sound wave and avoiding interference waves, which occur at certain points.

Third: The committee is not yet unanimous in opinion regarding the method of stimulating the fork, or rather of exciting it, and also the method of timing the fork. It was not definitely determined whether it is better to note the duration of the time during which the fork is heard, or to decide the degree of hearing by the distance from the ear at which the fork can be appreciated, using either a control or knowing the duration of the fork's vibration time. There is still lack of unanimity in the committee, and therefore it is impossible for us to make a very definite statement regarding this point. Neither method seems wholly accurate.

Fourth: The committee recommended that at first only three standard tests be considered, namely: The Weber, Schwabach and the Rinne. With reference to the first two tests, no decision was arrived at regarding which forks should be used. The committee thought one of the middle tones, perhaps a c^1 (256 double vibrations) or a somewhat lower one would do. For the Rinne test, a fork of middle tone, about c^1 (256 double vibrations) the exact pitch to be determined later, should be used. The time during which the fork is heard by air and bone conduction should be definitely designated. The construction of the forks ought to be such that the difference between air and bone conduction is about 30 to 40 seconds. When vibrating in the air alone, the fork should be heard approximately 100 seconds. This, of course, requires a rather expensive fork. Furthermore, the high limits should be determined either by means of the Galton whistle or the monochord. The latter is the only instrument whereby high tones can be tested by bone conduction.

Fifth: Regarding the forks recommended, there might be at first a minimum list, namely, a low fork of 32 double vibrations, a medium fork of 256 double vibrations and a high fork of 2048 double vibrations per second. Secondly, a desirable list consisting of five forks, namely, 32, 128, 256, 512 and 2048 double vibrations per second.

Sixth: The committee has conferred with expert manufacturers of standardized forks, and has also considered the feasibility or possibility of using rustless metal in the construction of the fork. When forks are nicked to prevent rusting, there is a disadvantage, first of all, that the nicking may interfere with the vibration, and secondly, when peeling occurs, adventitious sounds may be produced by the loose pieces of metal. On the other hand, if a fork is not nicked and is not kept properly oiled, rusting invariably occurs. Therefore we are greatly interested in the problem of obtaining rustless metal, but so far cannot state how soon and where these forks will be obtainable.

Seven: Attention is called to the fact that it is impossible to take the time to determine definitely what measures should be taken in so important a subject as the functional testing of hearing, but we desired to present the preliminary report and hoped for a full discussion on the floor, together with suggestions from members, either in person or in writing, in order to aid the committee in arriving at a definite conclusion.

Some of the members of your committee are in favor of using only c forks. Other members have been accustomed to

employing the Bezold A, (108 double vibrations) for the Weber and Schwabach test, an a₁ fork (435 double vibrations) for the Rinne test. Most of these forks have certain advantages hard to find in some of the other forks, but nevertheless they are not essential, and in case the others are employed, the same result will be had if the same care is taken in making the tests as outlined by Bezold in the use of the forks mentioned. In view of the fact that the members of the committee are not unanimous regarding the excitation of the forks or the determination of the hearing acuity, that is to say, whether the duration of the time the fork is heard or measuring the distance from the ear at which it is appreciated, it is impossible for us to make a definite recommendation. With reference to the recording of tuning fork and other functional testing, some members of the committee have for some time employed the acoumetric formula proposed and adopted at the International Medical Congress at Buda Pesth in 1909, and have found it useful and compact. Others employ the system used in the Vienna Clinic, the diagram being stamped upon the card and the findings noted thereon. There are others who employ the measure of the distance at which forks are heard, and express the degree of hearing in fractions, the denominator of which is the distance at which that particular fork on the average is heard by the normal individual and the numerator representing the distance heard by the patient who is being examined. Both the time and the distance method are fairly accurate if done carefully and systematically.

Your committee recommends that the suggestions made in the previous report be again emphasized. First: We suggest a minimum list of three forks, 32, 256 and 2048 double vibrations, and a desirable one of five forks ranging from 32 to 2048. For the high tones, either the Galton whistle or the Edelmann-Galton whistle or the monochord should be employed. Second: There should be a discussion regarding the method to be used in recording the tests. Third: Further effort should be made to investigate the manufacture of forks of rustless metal. Fourth: That the committee be continued for another year, and that an effort be made to solve the problems outlined, including experiments with reference to distance and timing in functional testing.

Respectfully submitted,

L. W. DEAN

G. W. MACKENZIE

E. G. GILL

ROBERT SONNENSCHN

It was moved, seconded and carried that the report be accepted and the committee continued.

THE PRESIDENT. Surgeon-General Ireland is in the room, and we would like to have a word from him.

SURGEON-GENERAL IRELAND. Mr. Chairman: This is certainly an unexpected pleasure to me. I just wanted to come and listen to your papers and meet some of my old associates. Of course I have nothing to say to a special meeting of this kind, but it is a great pleasure and honor to be asked to even stand before such a distinguished audience. (Applause)

THE SECRETARY. The Constitution and By-Laws have been submitted to a committee for revision. The members will receive copies of the changes proposed when the announcement is sent out for the meeting next year.

THE PRESIDENT. There is a committee from the American Medical Association, the Committee on Lye Legislation, of which Dr. Chevalier Jackson is chairman. If there is a member of that committee present we would be glad to hear from him. If not, I might say that Doctor Jackson has been working this matter up for a number of years, and has succeeded in getting through the Pennsylvania Legislature a law requiring the labeling of lye cans as poison. That is an important step. After the meeting of the Bronchoscopic Society, Dr. H. M. Taylor of Jacksonville, Florida, went home and succeeded in getting such a bill through the Florida Legislature without any trouble. Our endeavor now is to get it through every legislature in the country—a bill compelling manufacturers of lye to label it as poison. There has been some objection from lye manufacturers, and it seems to the committee that it is very necessary that every State should pass such a bill. I think there will be little trouble if each member will constitute himself a committee of one in his State and try to get it through your next Legislature. It might be well to have a committee from this society to act with Dr. Jackson's committee in behalf of this legislation.

It was moved by DR. A. A. HAYDEN that the Chair appoint a committee of three. Motion seconded and carried.

It was moved by DR. HORACE NEWHART that the Chair appoint a committee of five to be called the Committee on the Problems of the Hard of Hearing, this committee to work with a committee from the American Association of Organizations for the Deaf. Motion seconded by DR. ROBERT SONNENSCHNEIN, and carried. Meeting adjourned.

ALPHABETIC LIST

Honorary Fellows

- ELLIOTT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., London, W., Eng.
DUNDAS-GRANT, SIR JAMES,
148 Harley St., W. 1, London, Eng.
GRAY, ALBERT A.....Glasgow, Scotland
JOHNSON, LINDSAY..Britannia Bldg., West St., Durban, Natal
ELSCHNIG, PROF. A
Ferdinandstrasse, 10, Prague, Czecho-Slovakia
LAWFORD, J. B.,
99 Harley St., Cavendish Sq., W. London, Eng.
*LISTER, SIR WILLIAM T.,
24 Devonshire Place, W. 1, London, Eng.
STEPHENSON, SYDNEY,
24 Thayer St., Manchester Sq., London, Eng.
VAN DER HOEVE, PROF. J.,
Rijnsburgerweg, 6 A, Leiden, Holland

Life Members

- *DAYTON, W. L.....Funke Bldg., Lincoln, Neb., OALR
*FOSTER, HAL.....Altman Bldg., Kansas City, Mo., ALR
HOWE, LUCIEN....520 Delaware Ave., Buffalo, N. Y., Op.

Fellows

- *ABBOTT, W. J.....628 Union Bldg., Cleveland, O., OALR
ADAMS, A. L....323 West State St., Jacksonville, Ill., OALR
*ADAMS, C.....52 West State St., Trenton, N. J., OALR
ADAMS, DANIEL S.,
520 Hume-Mansur Bldg., Indianapolis, Ind., ALR
ADAMS, ELDRIDGE S..Moore Bldg., San Antonio, Tex., OALR
ADAMS, FRED'K W...Roosevelt Clinic, Seattle, Wash., OALR
AGNEW, FRED'K W.,
120 3rd Ave., S. E., Independence, Ia., OALR
AINSLEE, GEORGE,
606 Oregonian Bldg., Portland, Ore., OALR
ALBRIGHT, LT. P. M., M. C., U. S. Navy, U. S. S. Bridgeport,
Care of Postmaster, New York, N. Y., ALR
*ALDEN, ARTHUR M.,
3858 Westminster Place, St. Louis, Mo., ALR
*ALDERDYCE, W. W.....Nasby Blk., Toledo, O., OALR
ALEXANDER, GEORGE JAMES,
1831 Chestnut St., Philadelphia, Pa., ALR
ALKIRE, H. L.....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS. E.,
712 Waldheim Bldg., Kansas City, Mo., OALR

*Attended 1923 Meeting.

- ALLEN, G. H.....Mills Bldg., Topeka, Kan., OALR
 ALLEN, GEORGE V.,
 1434 Harrison Bldg., Topeka, Kan., OALR
 ALLEN, J. H.....Majestic Bldg., Denver, Colo., ALR
 ALLEN, L. P.....19 Jefferson Ave., Oshkosh, Wis., OALR
 ALLEN, S. E.....22 West 7th St., Cincinnati, O., ALR
 ALLPORT, F.....7 West Madison St., Chicago, Ill., OALR
 ALTER, F. W.....Colton Bldg., Toledo, O., OALR
 ALTRINGER, ARTHUR N.,
 1100 Rialto Bldg., Kansas City, Mo., ALR
 ALWAY, ROBERT D.,
 423 S. Lincoln St., Aberdeen, S.-D., OALR
 AMBERG, EMIL,
 David Whitney Bldg., Detroit, Mich., ALR
 ANDERSON, W. B...507 Main Ave., Brownwood, Tex., OALR
 *ANDREWS, A. H.....Reliance Bldg., Chicago, Ill., OALR
 ANDREWS, B. F...25 E. Washington St., Chicago, Ill., OALR
 *ANDREWS, H. D.....23 Allen St., Buffalo, N. Y., Op.
 ANDREWS, M. P....Dempsey Bldg., Manitowoc, Wis., OALR
 *ANNON, WALTER T..251 S. 17th St., Philadelphia, Pa., ALR
 APPLEMAN, L. F.,
 308 South 16th St., Philadelphia, Pa., Op.
 *ARBUCKLE, M. F.....Lister Bldg., St. Louis, Mo., ALR
 *ARNOLD, F. J.....182 Pearl St., Burlington, Vt., OALR
 *ARRELL, WILLIAM...317 Main, E., Hamilton, Ontario, OALR
 ARROWSMITH, H....170 Clinton St., Brooklyn, N. Y., ALR
 ASH, GARRETT G.....1 Main St., Bradford, Pa., OALR
 ASHLEY, T. W.....466 Park Ave., Kenosha, Wis., OALR
 ATKINSON, D. T.,
 1515 Russell Bldg., San Antonio, Tex., OALR
 *AUERBACH, J.,
 120 W. 86th St., New York City, N. Y., ALR
 AUFWASSER, H...1302 Greenup St., Covington, Ky., OALR
 AUTEN, F. E.,
 1st National Bank Bldg., Belleville, Ill., OALR
 AVERY, F. T.....357 W. 63rd St., Chicago, Ill., Op.LR
 AYRES, W. McL.....4 W. 7th St., Cincinnati, O., Op.
 AYNSWORTH, H. T.....Amicable Bldg., Waco, Tex., OALR
 *BAHN, CHAS. A.,
 722 Maison Blanche Annex, New Orleans, La., Op.
 BAILEY, F. W.....Security Bldg., Cedar Rapids, Ia., OALR
 BAILEY, H.....840 Landers Bldg., Springfield, Mo., OALR
 BAIRD, CHAS. G.....Beatrice, Neb., OALR
 BAKER, C. H.....Crapo Blk., Bay City, Mich., OALR
 BAKER, H. B.....59 Main St., Taunton, Mass., OALR
 *BALDWIN, KATE W.,
 1117 Spruce St., Philadelphia, Pa., ALR
 BALENTINE, P. L.,
 1524 Chestnut St., Philadelphia, Pa., Op.
 *BALL, J. MOORES.....4500 Olive St., St. Louis, Mo., Op.

- *BALL, M. V.....214 Penn Ave., W., Warren, Pa., OALR
- *BALLENGER, H. C.,
25 E. Washington St., Chicago, Ill., ALR
- BALLIN, M. J...108 E. 60th St., New York City, N. Y., ALR
- BANE, WM....330 Metropolitan Bldg., Denver, Colo., OALR
- BANE, W. C. 330 Metropolitan Bldg., Denver, Colo., OALR
- BANISTER, J. M.,
400 Brandeis Theater Bldg., Omaha, Neb., OALR
- *BARBER, F.....259 Alexander St., Rochester, N. Y., Op.
- BARCK, C.....Humboldt Bldg., St. Louis, Mo., Op.A
- BARDENHEIER, F. G. A....Frisco Bldg., St. Louis, Mo., ALR
- BARKAN, A.....77 Battery St., San Francisco, Cal., OALR
- BARKAN, H....Physicians Bldg., San Francisco, Cal., Op.
- BARKER, CHAS. B.,
224½ W. Oklahoma Ave., Guthrie, Okla., OALR
- *BARKER, O. G. A.,
Johnstown Trust Co., Johnstown, Pa., Op.
- BARNES, L. E...6860 South Halsted St., Chicago, Ill., OALR
- BARNHILL, J. F....Pennway Bldg., Indianapolis, Ind., ALR
- BARLOW, ROY A.,
461 Farmers Trust Bldg., South Bend, Ind., ALR
- BARON, F. S.....Peoples Bank Bldg., Zanesville, O., OALR
- BARTHOLOMEW, A. C.,
1020 Harrison St., Ft. Wayne, Ind., OALR
- BAUM, H. L.....Metropolitan Bldg., Denver, Colo., ALR
- BEACH, S. J.....704 Congress St., Portland, Me., OALR
- BEANE, GEORGE W.,
Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
- *BEATTIE, W. HENRY....252 Genesee St., Utica, N. Y., OALR
- *BEATTIE, ROBERT,
David Whitney Bldg., Detroit, Mich., OALR
- BEATTY, H. G.....150 E. Broad St., Columbus, O., OALR
- BEAUDOUX, H. A.,
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- BECK, F. L.....408 Hynds Bldg., Cheyenne, Wyo., OALR
- *BECK, J. C.....2551 N. Clark St., Chicago, Ill., ALR
- BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
- *BEDELL, A. J.....344 State St., Albany, N. Y., Op.A
- BEEBE, C. S.....Wells Bldg., Milwaukee, Wis., OALR
- *BEGGS, W. F.....2 Lombardy St., Albany, N. Y., OALR
- BEIL, J. WALLACE,
316 Argyle Bldg., Kansas City, Mo., OALR
- BELL, GEO. HUSTON,
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- BELL, M. H.,
First Natl. Bank Bldg., Vicksburg, Miss., OALR
- BELLIN, JOSEPH,
322 N. Washington St., Green Bay, Wis., OALR
- BELLOWS, GEORGE E...Rialto Bldg., Kansas City, Mo., Op.
- BEEM, I. F.....31 N. State St., Chicago, Ill., OALR
- *BENEDICT, W. L.....Mayo Clinic, Rochester, Minn., Op.

- BENETTO, FREDERICK R.,
172 Woolwich St., Guelph, Ont., Canada, OALR
- BENNETT, A. G.....26 Allen St., Buffalo, N. Y., Op.
- BENSON, GEORGE E.,
1111 Metropolitan Bank Bldg., Minneapolis, Minn., OALR
- BENTLEY, NEIL,
1161 David Whitney Bldg., Detroit, Mich., OALR
- *BERENS, CONRAD, JR.,
9 E. 46th St., New York City, N. Y., Op.
- *BERGERON, J. Z...104 S. Michigan Blvd., Chicago, Ill., ALR
- BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
- BERNSTEIN, E. J.....Kresge Bldg., Detroit, Mich., OALR
- BERRISFORD, PAUL D.,
812 Lowry Bldg., St. Paul, Minn., OALR
- BERRY, D. F...Hume-Mansur Bldg., Indianapolis, Ind., ALR
- BETTISON, D. L.....502 Wilson Bldg., Dallas, Tex., OALR
- BEYER, LOUIS J....449 Delaware Ave., Buffalo, N. Y., ALR
- *BIGELOW, NOLTON,
124 Waterman St., Providence, R. I., ALR
- BINGER, H. E.....1027 Lowry Bldg., St. Paul, Minn., OALR
- *BINKLEY, R. S.,
550 Fidelity Medical Bldg., Dayton, O., OALR
- BISHOP, C. W.,
616 LaSalle Bldg., Minneapolis, Minn., OALR
- *BLAAUW, E. E.....190 Ashland Ave., Buffalo, N. Y., OALR
- BLACK, M.....Metropolitan Bldg., Denver, Colo., Op.
- BLACK, N. M.....Wells Bldg., Milwaukee, Wis., Op.
- BLACK, W. D.....Metropolitan Bldg., St. Louis, Mo., OALR
- BLACKBURN, W. J....661 Reibold Bldg., Dayton, O., OALR
- *BLAIR, W. W...Diamond Bank Bldg., Pittsburgh, Pa., Op.
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- GROVE, W. E.....502 Wells Bldg., Milwaukee, Wis., OALR
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- *HARRIS, W. ROBIN...Exchange Bldg., Memphis, Tenn., ALR
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- HOWARD, J. W. Rialto Bldg., Kansas City, Mo., OALR
- HOWARD, LLOYD G. Council Bluffs, Iowa, OALR
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- *KING, CHARLES R.....471 Spitzer Bldg., Toledo, O., OALR
- KING, EDWARD....605 Traction Bldg., Cincinnati, O., ALR
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LIDIKAY, CHAS. J.....519 Portsmouth Bldg., OALR

MCCARTNEY, OLIVER P.....314 Portsmouth Bldg., OALR

SPAKE, L. B.....522 Minnesota Ave., ALR

LEAVENWORTH

HAAS, F. J.....Masonic Bldg., OALR

OTTAWA

NEIGHBORS, C. A.....Underwood Bldg., OALR

SCOTT, J. R.....201 S. Main St., OALR

PARSONS

LANDES, G. A.....110½ S. 18th St., OALR

PITTSBURG

GRAVES, WILBUR H.....Commerce Bldg., OALR

MUNSON, D. O.....Globe Bldg., OALR

STELLE, H. L.....Commerce Bldg., OALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR

ALLEN, G. H.....Mills Bldg., OALR

ALLEN, GEO. V.....Harrison Bldg., OALR

ESTERLY, D. E.....Mills Bldg.,

MAGEE, R. S.....Mills Bldg., Op.A

GEOGRAPHIC

WELLINGTON

SHULTS, E. J. G.....502 N. Washington St., OALR

WICHITA

CHENEY, JAMES W.....Schweiter Bldg., OALR

DORSEY, J. G.....201 N. Main St., OALR

GILLET, WILBUR G.....201 N. Main St., Op.

GSELL, J. F.....Beacon Bldg., OALR

HIGGINBOTHAM, T. L.....Belting Bldg., OALR

HUTCHINSON, O. G.....Schweiter Bldg., OALR

JOHNSON, J. H.....134 N. Main St., OALR

MAGGARD, D. I.....Beacon Bldg., OALR

PALMER, E. M.....Beacon Bldg., OALR

SEYDELL, E. M.....201 N. Main St., ALR

WEAVER, T. W.....201 N. Main St., Op.

WINFIELD

RALLS, C. T.....First National Bank Bldg., OALR

WILMER, F. M.....Castell Bldg., OALR

KENTUCKY

COVINGTON

AUFMWASSER, H.....1302 Greenup St., OALR

BLEDSE, R. W.....1005 Madison Ave., OALR

FULTON

COHN, SELDON.....416 Luke St., OALR

LEXINGTON

DEWEESE, CLARENCE.....120 W. Second St., Op.

OFFUT, W. N.....Security Bldg., OALR

STUCKY, J. A.....Fayette National Bank Bldg., OALR

STUCKY, W. S.....2nd and Upper Sts., OALR

LOUISVILLE

DABNEY, S. G.....Starks Bldg., OALR

DEAN, WALTER.....Francis Bldg., OALR

DULANEY, O.....717 Starks Bldg., OALR

HALL, G. C.....Gaulbert Bldg., OALR

HEITGER, J. D.....808 Francis Bldg., OALR

KELLY, C. W., JR.....Francis Bldg., OALR

LEDERMAN, ISAAC A.....Atherton Bldg., OALR

PFINGST, A. O.....Atherton Bldg., OALR

SHAFFER, J. J.....Starks Bldg., OALR

WOLFE, CLAUDE T.....Francis Bldg., OALR

NEWPORT

THOMASSON, W. J.....942 York St., OALR

PADUCAH

REYNOLDS, H. G.....City National Bank Bldg., Op.A

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....722 Maison Blanche Annex, Op.

BLUM, H. N.....Maison Blanche Bldg., Op.

FEINGOLD, MARCUS.....4206 St. Charles Ave., Op.
 HASPEL, D. M.....1214 Maison Blanche Bldg., ALR
 LANDFRIED, C. J. .607 Land Title and Guarantee Bldg., ALR
 LYNCH, R. C.....Maison Blanche Bldg., ALR
 SMITH, V. C.....Maison Blanche Bldg., Op
 TAQUINO, G. J.....Maison Blanche Bldg., ALR
 WEIL, A. L.....717 Maison Blanche Annex Bldg., ALR

SHREVEPORT

MOISE, A. B.....Commercial Bk. Bldg., ALR
 SCALES, J. L.....Commercial Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, O. W.....Augusta Trust Bldg., OALR

HOULTON

MITCHELL, FREDERICK W.....Fogg Bldg., OALR

PORTLAND

BEACH, S. J.....704 Congress St., OALR
 GILBERT, F. Y.....148 Park St., Op.AR
 HASKELL, A. D.....145 High St., Op.
 HOLT, E. E. JR.....723 Congress St., Op.A
 SPALDING, J. A.....627 Congress St., Op.A

MARYLAND

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., Op.
 FRIEDENWALD, HARRY.....1029 Madison Ave., Op.A
 GOLDBACH, L. J.....322 N. Charles St., OALR
 KEMLER, JOSEPH J.....1908 Eutaw Pl., OALR
 LOOPER, EDWARD A.....605 Park Ave., OALR
 PETERMAN, H. F.....518 N. Charles St., OALR
 TARUN, WILLIAM.....605 Park Ave., Op.A
 WOODS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
 ROBINSON, H. T.....Medical Bldg., OALR
 SHARRETT, GEORGE O.....119 Bedford St., OALR

MASSACHUSETTS

BOSTON

BRYANT, A. G.....502 Beacon St., ALR
 CARVILL, LIZZIE M.....101 Newbury St., Op.
 CHENERY, W. E.....377 Commonwealth Ave., ALR
 CHENEY, F. E.....64 Commonwealth Ave., Op.
 FREEDMAN, L. M.....419 Boylston St., ALR
 GREENWOOD, ALLEN.....82 Commonwealth Ave., Op.
 HURLEY, E. D.....419 Boylston St., Op.
 HURLEY, E. H.....419 Boylston St., Op.

INGLIS, H. J.....Bay State Bldg., ALR
 LANCASTER, W. B.....520 Commonwealth Ave., Op.
 LEMOINE, A. L.....233 Charles St., Op.
 LEWIS, J. P.....543 Boylston St., ALR
 MOSHER, H. P.....828 Beacon St., ALR
 RICE, G. B.....293 Commonwealth Ave., ALR
 ROWLAND, WILLIAM D.....220 Clarendon St., Op.
 SHANAHAN, T. J.....419 Boylston St., ALR
 STANDISH, MYLES.....51 Hereford St., Back Bay, Op.
 STEVENS, H. B.....522 Commonwealth Ave., Op.
 SUFFA, G. A.....Gulford Hall, Op.
 THOMPSON, P. H.....308 Commonwealth Ave., Op.
 TINGLEY, LOUISA P.....9 Massachusetts Ave., Op.
 TOBEY, G. L.....416 Marlborough St., ALR
 WELLS, D. W.....The Westminster, Op.
 WHITE, L. E.....390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....182 Concord St., OALR

GARDNER

BONE, H. D.....29 Parker St., OALR

HOLYOKE

HUSSEY, E. J.....276 High St., Op.LR

LAWRENCE

CONLON, F. A.....Bay State Bldg., OALR
 MERRILL, W. H.....Bay State Bldg., OALR

LYNN

COBB, C. M.....10 Nahant St., OALR
 JONES, E. W.....44 Atlanta St., Op.A
 O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....278 Union St., OALR

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....90 Main St., ALR
 DOW, F. E.....4 West St., Op.

PALMER

MOORE, G. A.....Bank Bldg., OALR

SALEM

HENNESSY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....	4 Chestnut St., OALR
CARLETON, RALPH.....	10 Temple St., Op.
FRELIGH, C. A.....	75 State St., ALR
IRWIN, V. J.....	389 Main St., OALR
IRWIN, V. J., JR.....	389 Main St., OALR

TAUNTON

BAKER, H. B.....	58 Main St., OALR
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WESTFIELD

CLARK, F. T.....	52 Broad St., OALR
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WORCESTER

CAHILL, J. W.....	390 Main St., OALR
CROSS, A. E.....	Slater Bldg., Op.
ESTABROOK, CHARLES.....	390 Main St., OALR

MICHIGAN

ANN ARBOR

SLOCUM, GEORGE.....	328 E. Huron St., Op.
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BATTLE CREEK

COLVER, B. N.....	182 Manchester St., OALR
FARNESWORTH, M. A.....	146 Greenwood St., OALR
HAUGHEY, WILFRID.....	24 W. Main St., OALR
SLEIGHT, R. D.....	Post Bldg., OALR
STEGMAN, L. B.....	Sanitarium, Op.
WENCKE, C. G.....	16 Wentworth Ct., OALR

BAY CITY

BAKER, C. H.....	Crapo Block, OALR
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DETROIT

AMBERG, EMIL.....	David Whitney Bldg., ALR
BEATTIE, ROBERT.....	David Whitney Bldg., OALR
BENTLEY, NEIL.....	1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....	Kresge Bldg., OALR
CAMPBELL, D. A.....	David Whitney Bldg., OALR
CAMPBELL, DON M.....	Peter Smith Bldg., OALR
CONNOR, RAY.....	1410 Stroh Bldg., OALR
DEFNET, WM. A.....	62 Adams St., W., ALR
GOUX, R. S.....	545 David Whitney Bldg., OALR
HARTZ, H. J.....	27 E. Adams St., ALR
MAIRE, L. E.....	Park Bldg., Op.A
McCLELLAND, CARL C.....	David Whitney Bldg., OALR
MERCER, R. E.....	David Whitney Bldg., LR
MINER, S. C.....	1564 Woodward Ave., ALR
ODELL, ANNA.....	32 Adams St., Op.AL
PARKER, WALTER R.....	David Whitney Bldg., Op.
PEIRCE, HORACE W.....	David Whitney Bldg., ALR
RENAUD, G. L.....	Fine Arts Bldg., OALR
SANDERSON, H. H.....	David Whitney Bldg., OALR
SHURLEY, B. R.....	62 W. Adams St., ALR
SIMPSON, H. L.....	David Whitney Bldg., ALR
WALKER, C. C.....	Woodward at Warren, Op.
WENDEL, JACOB S.....	David Whitney Bldg., ALR

GEOGRAPHIC

ESCANABA

BOYCE, WILLIAM B.....1019 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED304 Gilbert Blk. Bldg., Op.

GRANT, P. T.....Widdicomb Bldg., OALR

HUIZINGA, J. G.....Widdicomb Bldg., OALR

ROBERTSON, F. D.....Ashton Bldg., OALR

ROLLER, L. A.....122 Monroe St., OALR

WELSH, D. E.....Powers Theater Bldg., OALR

IONIA

SEDGWICK, O. W..... OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, G. E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B...Kalamazoo National Bank Bldg., OALR

GRANT, F. E. Kalamazoo National Bank Bldg., OALR

WILBUR, E. P.....Kalamazoo National Bank Bldg., OALR

MENOMINEE

ELWOOD, CALVIN R.....927 Main St., OALR

SAGINAW

McKINNEY, ALEXANDER R.....Bearinger Bldg., OALR

ROGERS, A. S.....105 S. Jefferson Ave., OALR

WATSON, ROY S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....206 Wilhelm Blk., OALR

MINNESOTA

DULUTH

BRIGGS, F. W.....Lyceum Bldg., ALR

COLLINS, HOMER.....New Jersey Bldg., OALR

TILDERQUIST, D. L.....7 E. Superior St., OALR

TURNBULL, F. M.....Providence Bldg., OALR

WINTER, JOHN A.....Fidelity Bldg., OALR

FERGUS FALLS

KITTLESon, THEODORE N.....101 Lincoln Ave., OALR

HIBBING

MORSMAN, L. W.....Congdon Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BEAUDOUX, H. A.....La Salle Bldg., Op.A

BENSON, GEO. E.....Metropolitan Bldg., OALR

BISHOP, C. W.....La Salle Bldg., OALR

BROWN, E. J.....Syndicate Blk., OALR

CAMP, W. E.....Donaldson Bldg., OALR
 CAMPBELL, ROBT. A.....La Salle Bldg., OALR
 CLARK, H. S.....Syndicate Blk., OALR
 DORGE, RICHARD I.....76 Seymour Ave., S. E., OALR
 HANSEN, E. W.....Donaldson Bldg., OALR
 HOWE, A. W.....4404 Pleasant Ave., OALR
 KERRICK, STANLEY E.....620 Syndicate Bldg., OALR
 LEE, J. W.....804 Besse Bldg., OALR
 LEWIS, J. D.....La Salle Bldg., OALR
 LITCHFIELD, J. T.....Phys. and Surgeons' Bldg., OALR
 LOOMIS, E. A.....Donaldson Bldg., OALR
 MACINE, J. S.....Donaldson Bldg., OALR
 MATTHEWS, JUSTUS.....Metropolitan Bldg., LR
 MORSE, J. A.....Donaldson Bldg., OALR
 MORTON, HOWARD McI.....Metropolitan Bk. Bldg., OALR
 MURRAY, WM. R.....Nicollet Clinic, OALR
 NEWHART, HORACE.....Donaldson Bldg., OALR
 OBERG, C. M.....Syndicate Blk., OALR
 PARKER, E. H.....La Salle Bldg., LR
 PATTERSON, W. E.....La Salle Bldg., OALR
 PHELPS, K. A.....La Salle Bldg., OALR
 PRATT, F. J., JR.....Metropolitan Bldg., OALR
 PRATT, J. A.....Metropolitan Bldg., OALR
 REYNOLDS, J. S.....La Salle Bldg., OALR
 SMITH, A. E.....Donaldson Bldg., OALR
 STROUT, E. S.....Donaldson Bldg., OALR
 STROUT, G. E.....Donaldson Bldg., OALR
 WATSON, J. A.....Phys. and Surg. Bldg., OALR
 WOOD, DOUGLAS.....Donaldson Bldg., OALR
 WRIGHT, C. D.....Metropolitan Bldg., Op.A

ROCHESTER

BENEDICT, W. L.....Mayo Clinic, Op.
 HEMPSTEAD, BERT E.....Mayo Clinic, ALR
 LILLIE, H. I.....Mayo Clinic, ALR
 LYONS, H. R.....Mayo Clinic, ALR
 NEW, G. B.....Mayo Clinic, LR
 PRANGEN, A. DE H.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
 BINGER, H. E.....Lowry Bldg., OALR
 BOECKMAN, EGIL.....Lowry Bldg., OALR
 BRAY, E. R.....934 Ashland Ave., OALR
 BROWN, JOHN C.....Lowry Bldg., OALR
 BURCH, F. E.....Hamm Bldg., A
 CONNOR, CHAS. E.....Lowry Bldg., OALR
 FOGARTY, CHAS. W.....1826 Summit Ave., OALR
 FULTON, JOHN E.....728 Lowry Bldg., OALR
 HOWARD, W. H.....137 N. Fairview Ave., OALR
 LARSEN, CARL E.....Lowry Bldg., OALR
 LEWIS, WILLIAM W.....836 Lowry Bldg., OALR

MALONEY, T. J.....Lowry Bldg., OALR
 NELSON, L. A.....Lowry Arcade, Op.A
 ROTHSCHILD, H. J.....Lowry Bldg., OALR
 SHELLMAN, J. L.....Lowry Bldg., OALR
 WHEELER, MERRITT W.....Lowry Bldg., OALR

MISSISSIPPI

MERIDIAN

GUTHRIE, J. M.....Rosenbaum Bldg., OALR

OXFORD

GUYTON, B. S.....Carter Bldg., OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., OALR
 EDWARDS, C. J.....1422 Wash., OALR
 HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

AURORA

O'DELL, TIMOTHY.....112 A Madison Ave., OALR

CARTHAGE

POST, WINIFRED B.....1380 S. Main St., OALR
 POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....311 Hitt St., OALR
 SNEED, C. M.....Guitar Bldg., OALR

JOPLIN

PIFER, J. D.....Frisco Bldg.

KANSAS CITY

ALLEN, CHAS. E.....Waldheim Bldg., OALR
 ALTRINGER, ARTHUR N.....Rialto Bldg., ALR
 BEIL, J. W.....Argyle Bldg., OALR
 BELLows, GEORGE E.....Rialto Bldg., Op.
 BLAKESLEY, T. S.....Lathrop Bldg., OALR
 BOURBON, OLIVER P.....Lathrop Bldg., OALR
 CURDY, R. J.....Rialto Bldg., Op.
 CURRAN, EDWIN J.....Waldheim Bldg., Op.
 DAVIS, HARRY B.....Lathrop Bldg., OALR
 FOSTER, HAL.....Altman Bldg., ALR
 GARRISON, B. E.....Argyle Bldg., OALR
 GOSNEY, C. W.....Lathrop Bldg., OALR
 HALL, EDW. P.....124 Hunter Ave., OALR
 HOWARD, J. W.....Rialto Bldg., OALR
 KEITH, W. E.....Waldheim Bldg., OALR
 KIMBERLIN, J. W.....Rialto Bldg., Op.
 LANSING, J. H.....Lathrop Bldg., ALR
 LEA, J. A.....Rialto Bldg., ALR
 LEONARD, A. C.....Bryant Bldg., OALR
 LICHTENBERG, J. S.....Rialto Bldg., Op

LORIE, ALVIN.....Commerce Bldg., ALR
 MAY, JAMES W.....800 Minnesota Ave., Op.
 MILLER, HUGH.....Chambers Bldg., OALR
 MOSS, H. E.....Rialto Bldg., OALR
 MOTT, J. S.....Argyle Bldg., OALR
 MYERS, J. L.....Lathrop Bldg., OALR
 McALESTER, A. W., JR.....Bryant Bldg., Op.
 McCARTY, V. W.....Rialto Bldg., ALR
 PAINTER, A. M.....Sharp Bldg., ALR
 PATTERSON, J. M.....Bryant Bldg., OALR
 ROBERTS, S. E.....Waldheim Bldg., ALR
 RUSSELL, E. L.....Argyle Bldg., OALR
 SCHUTZ, W. H.....Bryant Bldg., OALR
 SELLERS, L. M.....Argyle Bldg., ALR
 SHOEMAKER, S. A.....U. S. Pub. Ser. Hosp., OALR
 SHUMATE, D. L.....Shukert Bldg., OALR
 THOMASON, H. E.....Rialto Bldg., OALR
 TUREMAN, H. G.....Rialto Bldg., OALR
 YAZEL, H. E.....Grand Ave. Temple, OALR

MOBERLY

DUTTON, C. K..... OALR

NEVADA

McLEMORE, TIPTON.....H. C. Moore Bldg., OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....Logan Bldg., OALR
 KENNEY, W. L.....Corby Forsee Bldg., OALR
 MINTON, W. H.....Bartlett Bldg., OALR
 PROUD, W. C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....3858 Westminster Place, ALR
 ARBUCKLE, M. F.....Lister Bldg., ALR
 BALL, J. MOORES.....4500 Olive St., Op.
 BARCK, CARL.....Humboldt Bldg., Op.A
 BARDENHEIER, F. G. A.....Frisco Bldg., Op.A
 BLACK, W. D.....Metropolitan Bldg., OALR
 BRYAN, W. M. C.....University Club Bldg., ALR
 BURNS, S. S.....Humboldt Bldg., ALR
 CHARLES, J. W.....Humboldt Bldg., Op.
 DONNELL, NEWMAN R.....Metropolitan Bldg., Op.
 DYER, CLYDE P.....Metropolitan Bldg., Op.
 EWING, ARTHUR E.....Metropolitan Bldg., Op.
 GOLDSTEIN, M. A.....3858 Westminster Pl., ALR
 GREEN, JOHN, JR.....Metropolitan Bldg., Op.
 GUGGENHEIM, L. K.....Carleton Bldg., ALR
 GUNDELACH, C. A.....825 University Club Bldg., ALR
 HARDESTY, J. F.....Humboldt Bldg., Op.
 HARDY, W. F.....Metropolitan Bldg., Op.
 HIGBEE, E. H.....Metropolitan Bldg., Op.
 HOURN, G. E.....Univ. Club Bldg., ALR
 JACOBS, M. W.....Carleton Bldg., Op.

JENNINGS, J. E.....Carleton Bldg., OALR
 LAMB, HARVEY.....Metropolitan Bldg., Op.
 LEAVY, C. A.....Metropolitan Bldg., ALR
 LOEB, H. W.....Humboldt Bldg., ALR
 LUEDDE, W. H.....Metropolitan Bldg., Op.
 LYMAN, HARRY W.....Carleton Bldg., ALR
 PFINGSTEN, C. F.....508 N. Grand Ave., ALR
 POST, LAWRENCE.....Metropolitan Bldg., Op.
 POST, MARTIN H.....Metropolitan Bldg., Op.
 PROETZ, ARTHUR W.....University Club Bldg., ALR
 REIM, HUGO.....Metropolitan Bldg., Op.
 SAUER, W. E.....Humboldt Bldg., ALR
 SCHOLZ, R. P.....Metropolitan Bldg., ALR
 SCHWARTZ, F. A.....Metropolitan Bldg., Op.
 SHAHAN, W. E.....Metropolitan Bldg., Op.
 SHOEMAKER, W. A.....Carleton Bldg., Op.
 SLUDER, GREENFIELD.....3542 Washington Ave., ALR
 TOOKER, C. W.....Carleton Bldg., Op.
 WESTLAKE, S. V.....Humboldt Bldg., ALR
 WIENER, MEYER.....Carleton Bldg., Op.
 WOODRUFF, F. E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, J. G.....Ilgenfritz Bldg., OALR
 TITSWORTH, GUY.....111 W. 4th St., Op.

SPRINGFIELD

BAILEY, H.....840 Landers Bldg., OALR
 COFFELT, THEODORE A.....Woodruff Bldg., OALR
 KLINGER, THOS. O.....Landers Bldg., OALR

TRENTON

VAUGHN, G. E.....Trenton Trust Bldg., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DAYTON, G. O.....Lewisohn Bldg., OALR
 DONAVAN, J. A.....Phoenix Bldg., OALR
 MORSE, A. W.....Phoenix Bldg., OALR
 POTTER, PETER.....Granate and Alaska Sts., OALR

GREAT FALLS

COULTER, C. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

DAVIS, FRANK C.....ALR

ROUNDUP

LEWIS, G. A.....29 Main St., OALR

NEBRASKA

BEATRICE

BAIRD, CHARLES G..... OALR
TUCKER, J. C..... OALR

COLUMBUS

CAMPBELL, C. H..... Columbus State Bk. Bldg., OALR

KEARNEY

FOX, CHAS. H..... 2116 Central Ave., OALR

LINCOLN

BROOKS, E. B..... Terminal Bldg., OALR
COOK, S. E..... Richards Block, OALR
CURTIS, W. L..... 612 Security Mutual Bank Bldg., OALR
DAYTON, W. L..... Funke Bldg., OALR
FURGASON, A. P..... Richards Block, OALR
HOMPES, J. J..... Security Mutual Bldg., OALR
SANDERSON, DAVID D..... Funke Bldg., OALR
WILLIAMS, J. P..... Funke Bldg., OALR
WOODWARD, J. M..... Richards Block
ZEMER, S. G..... Security Mutual Bldg., OALR

NORFOLK

GADBOIS, A. E..... 421 Norfolk Ave., OALR
NELSON, LLOYD L..... OALR

OMAHA

BANISTER, J. M..... Brandeis Theater Bldg., OALR
CALLFAS, W. F..... Brandeis Bldg., ALR
FAIRCHILD, NORA M..... 600 Brandeis Theater Bldg., Op.
GIFFORD, HAROLD..... Brandeis Bldg., Op.A
GIFFORD, SANFORD R..... 702 Brandeis Theater Bldg., Op.
HANEY, W. P..... Brandeis Theater Bldg., OALR
ISAAC, DAVID..... Brandeis Theater Bldg., OALR
KNOVDE, A. R..... Omaha National Bank Bldg., ALR
KULLY, B. M..... 631 City Natl. Bk. Bldg., ALR
LEMERE, H. B..... Brandeis Bldg., OALR
OWEN, F. S..... Brandeis Bldg., OALR
PATTON, J. M..... Brandeis Bldg., OALR
POTTER, GEORGE B..... 402 City Natl. Bk. Bldg., OALR
POTTS, J. B..... Brandeis Bldg., AL
RUBENTHAL, CLARENCE..... Brandeis Bldg., OALR
TUCKER, J. C..... Brandeis Bldg., OALR
UREN, C. T..... Omaha National Bank Bldg., OALR
WHERRY, W. P..... Bankers Reserve Life Bldg., OALR

NEW HAMPSHIRE

HANOVER

CARLETON, E. H..... ALR

NASHUA

NUTTER, C. F..... 16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois St., ALR

MCGIVERN, CHAS. S.....805 Pacific Ave., ALR

STICKNEY, O. D.....92 Pacific Ave., ALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

CHAMBERS, T. R.....15 Exchange Pl., OALR

PYLE, WALLACE.....18th and Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CHATTIN, J. F.....671 Broad St., Op.

HURF, J. W.....71 Washington St., OALR

O'CONNOR, F. O.....671 Broad St., OALR

ORTON, H. B.....671 Broad St., ALR

QUINBY, W. O'G.....14 James St., OALR

SHERMAN, E. S.....Wiss Bldg., OALR

ZEHNDER, C. A.....180 Fairmount Ave., OALR

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

TRENTON

ADAMS, CHARLES.....52 W. State St., OALR

CRANE, J. WELLINGTON.....128 Third St., ALR

WEST HOBOKEN

SACCO, A. G.....440 Clinton Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHEMER, HARRISON L.....521 Luna Blvd., OALR

NEW YORK

ALBANY

BEDELL, A. J.....344 State St., Op.A

BEGGS, W. F.....2 Lombardy St., OALR

DOWLING, J. I.....116 Washington Ave., OALR

JUDGE, H. V.....228 State St., Op.

BINGHAMTON

WATSON, H. D.....151 Front St., OALR

BROOKLYN

ARROWSMITH, HERBERT.....170 Clinton St., ALR

HANCOCK, J. C.....135 Cambridge Pl., Op.

LOMBARDO, MELCHIORE.....186 Meserole St., Op.

McCLELLAND, L. A.....78 McDonough St., ALR
 MYERSON, MERVIN C.....184 Clinton St., ALR

BUFFALO

ANDREWS, H. D.....23 Allen St., Op.
 BENNETT, A. G.....26 Allen St., Op.
 BEYER, LOUIS J.....449 Delaware Ave., ALR
 BLAAUW, E. E.....190 Ashland Ave., OALR
 BOZER, HERRMANN, E.....438 Delaware Ave., ALR
 BROWN, C. M.....510 Delaware Ave., ALR
 COTT, C. C.....483 Delaware Ave., ALR
 COWPER, H. W.....543 Franklin St., Op.
 EDSON, RAY.....560 Delaware Ave., Op.
 FAIRBAIRN, JOHN.....503 Delaware Ave., ALR
 FLAGG, J. D.....473 Virginia Ave., Op.A
 FRANCIS, L. M.....636 Delaware Ave., Op.
 GLOSSER, HERBERT H.....448 Franklin St., OALR
 HEALEY, J. F.....503 Delaware Ave., ALR
 HOWE, LUCIEN.....520 Delaware Ave., Op.
 HUBBARD, A. E.....372 Franklin St., Op.
 LEWIS, F. P.....454 Franklin St., Op.
 MARCH, CLARA A.....465 Ashland Ave., Op.
 PHILLIPS, W. L.....759 Richmond Ave., Op.
 RENNER, W. S.....341 Linwood Ave., ALR
 SATTERLEE, R. H.....187 Delaware Ave., Op.
 SERNOFFSKY, I.....37 Allen St., Op.
 STARR, E. G.....523 Delaware Ave., Op.
 WEED, HARRY M.....196 Lincoln Ave., Op.

DUNKIRK

JACKLE, A. F.....63 E. 4th St., Op.

GENEVA

SPENGLER, J. A.....423 Main St., OALR

ITHACA

BULL, E. L.....124 E. State St., OALR
 KIRKENDELL, J. S.....315 N. Aurora, OALR
 WILSON, R. C.....208 E. State St., OALR

JAMESTOWN

HOTCHKISS, W. W.195 Forest Ave., OALR
 REGER, H. S.....New Wellman Bldg., OALR

KENMORE

WURTZ, W. J. M.....2808 Delaware Ave., ALR

LIBERTY

DWORETSKY, JULIUS.....7 Law St., ALR

LOCKPORT

RINGUEBERG, EUGENE.....15 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., OALR

NEW YORK CITY

ALLBRIGHT, LT. P. M., M. C., U. S. Navy, U. S. S. Bridgeport, Care Postmaster, New York, N. Y.,	ALR
AUERBACH, JULIUS.....	120 W 86th St., ALR
BALLIN, M. J.....	108 E. 60th St., ALR
BELL, GEO. HUSTON.....	40 E. 41st St., Op.
BERENS, CONRAD, JR.....	9 E. 46th St., Op.
BRUDER, JOSEPH.....	230 W. 79th St., OALR
CARTER, W. W.....	2 W. 67th St., ALR
COHEN, MARTIN.....	1 W. 85th St., OALR
COHN, FELIX.....	31 E. 60th St., ALR
DAVIS, A. E.....	47 E. 57th St., Op.
DAVIS, G. E.....	42 W. 77th St., OALR
DIXON, G. S.....	40 E. 41st St., R
DUANE, ALEXANDER.....	139 E. 37th St., Op.
FORBES, H. H.....	40 E. 41st St., ALR
FREUDENTHAL, WOLFF.....	24 W. 88th St., ALR
FRIDENBERG, P. H.....	38 W. 59th St., OALR
FRIEDMAN, DAVID.....	44 W. 77th St., ALR
GATEWOOD, W. L.....	15 E. 48th St., ALR
GLOGAU, OTTO.....	64 E. 91st St., OALR
GOLDMAN, HARRY G.....	58 W. 58th St., ALR
GOTTLIEB, M. J.....	814 West End Ave., ALR
GRUSHLOW, ISRAEL.....	271 Central Park, W., ALR
GUTTMAN, J.....	616 Madison Ave., OALR
HASKIN, W. H.....	40 E. 41st St., ALR
HAYS, H. N.....	22 W. 74th St., ALR
HETRICK, LLEWELLYN E.....	30 W. 48th St., ALR
HOPKINS, W. E.....	515 Park Ave., OALR
HURD, L. M.....	39 E. 50th St., ALR
IMPERATORI, C. J.....	17 E. 38th St., ALR
INGERMAN, SERGIUS.....	Hotel Brunswick, OALR
IRWIN, F. N.....	114 E. 54th St., OALR
JACOBS, S. M.....	375 E. 149th St., OALR
JARECKY, HERMAN.....	138 W. 86th St., OALR
JOHNSON, THOMAS H.....	30 W. 59th St., Op.
JUDD, H. B.....	47 E. 57th St., ALR
KERRISON, P. D.....	58 W. 56th St., ALR
KEY, B. W.....	7 W. 49th St., Op.
KING, J. J.....	40 E. 41st St., ALR
KNAPP, ARNOLD.....	10 E. 54th St., Op.
KOPETZKY, S. J.....	51 W. 73rd St., ALR
KRUG, E. F.....	12 W. 44th St., OALR
LA VIGNE, A. A.....	11 E. 48th St., OALR
LUBMAN, MAX.....	616 Madison Ave., ALR
MAY, C. H.....	698 Madison Ave., Op.
MAYBAUM, J. E.....	17 E. 38th St., ALR
MAYER, EMIL.....	40 E. 41st St., ALR
MCCOY, JOHN.....	157 W. 73rd St., ALR
MEIERHOF, E. L.....	1140 Madison Ave., OALR
MITTENDORF, A. D.....	399 Park Ave., Op.

MITTENDORF, W. K.....115 E. 53rd St., Op.A
 MORTIMER, W. GOLDEN.....58 Central Park, W., OALR
 NISSELSON, MAX.....616 Madison Ave., OALR
 OPENHEIMER, SEYMOUR.....45 E. 60th St., OALR
 PAYNE, S. M.....542 Fifth Ave., OALR
 PHILLIPS, W. C.....40 W. 47th St., ALR
 REESE, R. G.....50 W. 52nd St., Op.
 SCHILLER, A. N.....225 West End Ave., ALR
 SOMBERG, JOSEPH S.....45 E. 60th St., Op.
 STURGES, L. F.....128 W. 87th St., LR
 VOORHEES, IRVING W.....14 Central Park, W., ALR
 WEEKS, JOHN E.....46 E. 57th St., Op.
 WEEKS, WEBB W.....20 E. 53rd St., Op.
 WHEELER, J. M.....30 W. 59th St., Op.
 WIENER, ALFRED.....550 Park Ave., OALR
 YANKAUER, SIDNEY.....616 Madison Ave., ALR

NIAGARA FALLS

PRICE, N. W.....445 Third St., OALR
 ROOKER, A. L.....1347 Michigan Ave., ALR

OLEAN

TINDOLPH, LEA W.....First Natl. Bk. Bldg., OALR

ONEIDA

CROCKETT, R. L.....Op.

POUGHKEEPSIE

KRIEGER, W. A.....36 Market St., OALR

ROCHESTER

BARBER, FRANK.....259 Alexander St., Op.
 CARROLL, G. G.....614 Main St., W., OALR
 CLARK, L. H.....337 Monroe Ave., OALR
 LERNER, MACY L.....332 Park Ave., Op.
 McDOWELL, N. D.....275 Alexander St., OALR
 MORRIS, A. G.....41 Gibbs St., OALR
 SHAPERO, I. M.....365 East Ave., OALR

SYRACUSE

BROWN, M. G.....802 University Bldg., ALR
 BRITTEN, G. S.....University Blk., OALR
 BRUST, H. O.....720 S. Crouse Ave., ALR
 FOWLER, S. R.....University Bldg., ALR
 KLINE, H. G.....University Blk., ALR

TROY

SULZMAN, F. M.....1831 Fifth Ave., OALR

UTICA

BEATTIE, W. HENRY.....252 Genesee St., OALR

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....73 Haywood St., OALR
 GREENE, JOSEPH B.....Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P.....511 Independence Bldg., OALR
 PEELER, C. N.....W. 7th St., OALR
 SLOAN, HENRY L.....Independence Bldg., Op.
 WHISNANT, A. M.....Realty Bldg., OALR

GREENSBORO

REAVES, W. P.....117 W. Sycamore St., OALR

NEW BERN

DANIELS, R. L.....Elks' Temple, OALR

RALEIGH

WRIGHT, J. B.....Citizens Natl. Bk. Bldg., OALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., OALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....De Lendrecie Blk., OALR
 RINDLAUB, J. H.....De Lendrecie Blk., OALR
 RINDLAUB, M. P., Jr.....De Lendrecie Blk., OALR

MINOT

McCONNELL, A. D.....Colcord Bldg., OALR

WAHPETON

LANCASTER, WILSON.....310 Dakota Ave., OALR
 RICE, C. P.....Sterns Bldg., OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR
 MOORE, T. K.....Hamilton Bldg., OALR
 STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....437 E. Market St., OAR

BELLEFONTAINE

BUTLER, ROBERT H.....227 W. Columbus Ave., OALR
 HARBERT, J. P.....Canby Blk., OALR

CANTON

SCHILD, E. H.....9th and Cleveland, Op.A

CINCINNATI

ALLEN, S. E.....22 W. 7th St., OALR
 AYKES, W. McL.....4 W. 7th St., Op.
 HANS, C. L.....2900 Grasselli Ave., OALR
 HARRIS, W. C.....Groton Bldg., ALR

HEFLEBOWER, R. C.....	The Lancaster, OALR
HINNEN, G. A.....	1343 Delta Ave., OALR
IGLAUER, SAMUEL.....	Livingston Bldg., ALR
KING, C.....	Union Central Bldg., Op.
KING, EDWARD.....	605 Traction Bldg., ALR
LAMB, F. W.....	7th and Vine Sts., OALR
MITHOEFER, WILLIAM.....	19 W. 7th St., ALR
MURPHY, W. E.....	7th and Race Sts., OALR
PHINNEY, F. D.....	22 W. 7th St., OALR
RAY, VICTOR.....	19 W. 7th St., OALR
SIEGEL, F. X.....	2700 Union Central Bldg., OALR
STANBERRY, HENRY.....	Provident Bk. Bldg., Op.AR
STEVENSON, ROBERT.....	22 W. 7th St., ALR
STEWART, T. M.....	Union Trust Bldg., OALR
STRICKER, LOUIS.....	Groton Bldg., Op.
TANGEMAN, H. F.....	Central Bldg., ALR
THOMSON, E. H.....	13 W. 7th St., OALR
URNER, M. H.....	Union Central Life Bldg., OALR
VAIL, D. T.....	24 E. 8th St., Op.

CLEVELAND

ABBOTT, W. J.....	628 Union Bldg., OALR
BRUNER, ABRAM B.....	Guardian Bldg., Op.
BRUNER, W. E.....	Guardian Bldg., Op.
BURKE, T. A.....	Rose Bldg., OALR
COGAN, J. E.....	Rose Bldg., OALR
CUTLER, FRANKLIN E.....	1025 Schofield Bldg., ALR
FORSYTHE, S. T.....	1011 Euclid Ave., OALR
INGERSOLL, J. M.....	Osborn Bldg., ALR
KENDALL, M. R.....	Osborn Bldg., ALR
LARGE, S. H.....	2323 Prospect Ave., ALR
LENKER, J. N.....	Osborn Bldg., ALR
METZ, R. B.....	Guardian Bldg., Op.
METZENBAUM, MYRON.....	Rose Bldg., ALR
MONSON, S. H.....	Ainsfield Bldg., OALR
MCDONALD, C. L.....	Ainsfield Bldg., OALR
NELSON, C. F.....	Schofield Bldg., OALR
PITKIN, C. E.....	Osborn Bldg., ALR
PRENDERGAST, D.....	1110 Euclid Ave., OALR
QUITTNER, S. S.....	3912 Prospect Ave., OALR
RUST, E. G.....	Hanna Bldg., Op.A
SHACKELTON, W. E.....	2323 Prospect Ave., Op.
SHIRAS, H. H.....	Guardian Bldg., Op.
SIMONDS, O. F.....	Rose Bldg., Op.
STOTTER, A. L.....	1148 Euclid Ave., OALR
STOTTER, JAMES.....	1148 Euclid Ave., OALR
TRIPP, I. C.....	Rose Bldg., OALR
TUCKERMAN, W. C.....	Osborn Bldg., Op.
TUCKERMAN, W. H.....	Osborn Bldg., OALR
WAUGH, J. M.,	

Cleveland Clinic, Euclid Ave. at 93rd St., ALR
 WOLFENSTEIN, LEO.....Rose Bldg., Op.

COLUMBUS

BEATTY, H. G.....	150 E. Broad St.,	OALR
BROWN, J. E.....	239 E. Town St.,	OALR
CLARK, I. G.....	188 E. State St.,	OALR
HAUER, A. M.....	327 E. State St.,	OALR
MEANS, C. S.....	131 E. State St.,	OALR
PROUT, A. W.....	370 E. Town St.,	OALR
SANOR, D. G.....	206 E. State St.,	OALR
SCHAEFFER, G. C.....	216 E. State St.,	OALR
SILBERNAGEL, C. E.....	283 E. State St.,	OALR
SULZER, G. A.....	200 E. State St.,	OALR
THOMAS, F. W.....	74 S. 5th St.,	OALR
TIMBERMAN, ANDREW.....	112 E. Broad St.,	OALR
WOLFE, A. C.....	350 E. State St.,	ALR
WRIGHT, J. W.....	Central National Bank Bldg.,	Op.

DAYTON

BINKLEY, R. S.....	Fidelity Med. Bldg.,	OALR
BLACKBURN, W. J.....	Reibold Bldg.,	OALR
BONNER, HORACE.....	Fidelity Med. Bldg.,	OALR
DUTROW, H. V.....	1040 Fidelity Medical Bldg.,	OALR
FARMER, A. G.....	Fidelity Med. Bldg.,	OALR
FOUTS, JOHN D.....	Fidelity Medical Bldg.,	OALR
HARRIS, H. B.....	Fidelity Med. Bldg.,	OALR
MILLETTE, J. W.....	Reibold Bldg.,	OALR
WEBSTER, R. M.....	Reibold Bldg.,	OALR

ELYRIA

GILL, GEORGE.....	146 Middle Ave.,	OALR
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IRONTON

DUNN, O. B.....	Fourth and Railroad Sts.	
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LIMA

STUEBER, F. G.....	Main and North Sts.,	OALR
STUEBER, PAUL J.....	209 W. North St.,	OALR

LORAIN

MONOSMITH, O. B.....	424 Broadway,	OALR
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MANSFIELD

GARBER, J. M.....	48 Park Ave.,	OALR
WISE, R. C.....	News Bldg.,	OALR

MARIETTA

SAUER, W. W.....	Box 416,	OALR
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MARION

BRICKLEY, D. W.....	196 W. Center St.,	OALR
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MIDDLETOWN

WILLIAMS, WALTER H.....		OALR
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NEWARK

HATCH, C. B.....	3 W. Church St.,	OALR
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SALEM

HILL, A. J.....	301 Citizens Natl. Bk. Bldg.,	OALR
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SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
 HARTLEY, F. A.....Fairbanks Bldg., OALR
 HOGUE, D. W.....Fairbanks Bldg., OALR
 MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, W. W.....Nasby Bldg., OALR
 ALDER, F. W.....Colton Bldg., OALR
 DENMAN, IRA O.....Ohio Bldg., OALR
 HOBART, BERTHA K.....Produce Exchange Bldg., OALR
 JACOBI, FRANK.....Colton Bldg., OALR
 KELLER, T. F.....The Spitzer, OALR
 KING, CHARLES R.....Spitzer Bldg., OALR
 LUKENS, CHARLES.....218 Michigan Ave., OALR
 SNYDER, W. H.....211 Ontario St., OALR
 STEINFELD, A. L.....234 Michigan St., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
 SHIELDS, L.....Allen Bldg., ALR

YOUNGSTOWN

GIBSON, R. D.....Dollar Savings and Trust Bldg., OALR
 HARTZELL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

BARON, F. S.....Peoples Bank Bldg., OALR
 CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Central Natl. Bk. Bldg., OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Oklahoma Ave., OALR

MCALISTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....706 1st Natl. Bank Bldg., OALR
 FERGUSON, E. I.....607 1st Natl. Bank Bldg., OALR
 GUTHRIE, A. L.....Amer. Natl. Bank Bldg., OALR
 MCHENRY, D. D.....Colcord Bldg., OALR

NEWTON, L. A.....Colcord Bldg., OALR
 TODD, H. C.....Colcord Bldg., OALR

TULSA

BRASWELL, C. J.....726 Mayo Bldg., OALR
 ROTH, A. W.....Palace Bldg., OALR

OREGON

LA GRANDE

BOUVY, HARRY.....New Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Oregonian Bldg., OALR
 BRUERE, G. E.....Journal Bldg., OALR
 DAVIS, RALPH F.....Selling Bldg., OALR
 FENTON, RALPH A.....616 Journal Bldg., OALR
 HENTON, GEORGE EARL.....Morgan Bldg., OALR
 JOHNSTON, WILSON.....Stevens Bldg., OALR
 KIEHLE, F. A.....Corbett Bldg., OALR
 KISTNER, F. B.....Stevens Bldg., ALR
 MCCOOL, JOSEPH L.....Stevens Bldg., Op.

PENNSYLVANIA

ALTOONA

GLOVER, S. P.....1118, 12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., OALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....I. O. O. F. Temple, OALR
 HAZLETT, L. R.....117 S. Main St., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR
 STAHLMAN, F. C.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

ERIE

DENNIS, D. N.....221 W. 9th St., Op.A
 DENNIS, E. P.....311 W. 10th St., OALR
 DUNN, I. J.....Masonic Temple, OALR
 FUST, J. H. E.....234 W. 6th St., OALR
 HEARD, C. F.....Masonic Temple, OALR
 JACKSON, J. D.....2324 W. 8th St.
 RUSSELL, J. A.....206 Masonic Temple, ALR
 SCHLINDWEIN, G. W.....138 W. 9th St., OALR
 SHREVE, O. M.....162 W. 8th St., OALR
 WRIGHT, KATHERINE L.....247 W. 8th St., ALR

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

McKEE, C. W.....Coulter Bldg., OALR

HARRISBURG

DAILEY, G. L.....713 N. 3rd St., OALR

FARNSLER, H. H.....1438 Market St., OALR

PARK, J. W.....32 N. 2d St., OALR

REBUCK, C. S.....412 N. 3d St., OALR

HAZELTON

REICHE, O. C.....328 W. Broad St., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....Johnstown Trust Bldg., Op.

HARRIS, CLARENCE M.....Johnstown Trust Bldg., OALR

RUSH, CALVIN C.....342 Main St., OALR

MIDDLETOWN

GEORGE, H. W.....19 N. Union St., Op.

NEW BRIGHTON

NORTON, ROY R.....Masonic Bldg., OALR

NEW KENSINGTON

McCONNELL, T. E.....Alter Bldg., OALR

STEIM, JOSEPH M.....1st Natl. Bk. Bldg., Op.

PHILADELPHIA

ALEXANDER, GEORGE J.....1831 Chestnut St., ALR

ANNON, WALTER T.....251 S 17th St., ALR

APPLEMAN, L. F.....308 S. 16th St., Op.

BALDWIN, KATE W.....1117 Spruce St., ALR

BALENTINE, P. L.....1524 Chestnut St., Op.

BOEHRINGER, H. W.....1811 S. 22nd St., Op.

BRINKERHOFF, NELSON M.....1831 Chestnut St., Op.

BROWN, S. H.....1901 Mt. Vernon St., Op.

BRUMM, SETH A.....Stock Exchange Bldg., ALR

BUTLER, MARGARET F.....1831 Chestnut St., ALR

BUTT, M. M.....2045 Chestnut St., Op.

CLERF, LOUIS H.....128 S. 10th St., ALR

COATES, G. M.....1736 Pine St., ALR

COHEN, SAMUEL.....1626 Spruce St., ALR

CRAMPTON, G. S.....1700 Walnut St., Op.

CREIGHTON, W. J.....1905 Chestnut St., Op.

CROSKEY, J. W.....1909 Chestnut St., OALR

DAVIS, J. L.....135 S. 18th St., ALR

DEICHLER, LYN WALTER.....2028 Chestnut St., OALR

DINTENFASS, HENRY.....1714 Pine St., ALR

EARL, HARRY D.....3717 Spruce St., ALR

ERSNER, M. S.....1729 Pine St., ALR

FELT, C. L.....2007 Chestnut St., ALR

FOX, L. W.....	303 S. 17th St., Op.
GLEASON, E. B.....	2033 Chestnut St., ALR
GODDARD, H. M.....	1531 Spruce St., ALR
GRISCOM, J. M.....	1925 Chestnut St., Op.
HAWMAN, E. G.....	4051 Broad St., OALR
HEED, C. R.....	1205 Spruce St., Op.
HERBERT, A. W.....	119 E. Lehigh Ave., OALR
HOLLOWAY, T. B.....	1819 Chestnut St., Op.
HUSIK, DAVID N.....	1610 Spruce St., ALR
JACKSON, C.....	128 S. 10th St., L
KAUFMAN, A. S.....	1923 Spruce St., ALR
KEELER, J. C.....	254 S. 16th St., ALR
KRAUSS, FREDERICK.....	1701 Chestnut St., OALR
LAESSLE, H. A.....	The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....	Medical Arts Bldg., Op.
LEWIS, FIELDING O.....	259 S. 17th St., ALR
LOFTUS, J. E.....	605 Medical Arts Bldg., ALR
LOVE, LOUIS F.....	1305 Locust St., Op.
LUKENS, R. McD.....	1308 Hunting Park Ave., ALR
MACFARLIN, DOUGLAS.....	1805 Chestnut St., OALR
MACKENZIE, ALICE V.....	1724 Spruce St., OALR
MACKENZIE, GEORGE W.....	1724 Spruce St., ALR
MILLER, E. B.....	2008 Walnut St., Op.
MITCHELL, E. K.....	704 Lehigh Ave., ALR
MOORE, W. F.....	255 S. 16th St., ALR
O'REILLY, C. A.....	1901 Chestnut St., ALR
PETER, LUTHER C.....	1529 Spruce St., Op.
PONTIUS, P. J.....	1831 Chestnut St., OALR
POSEY, WM. CAMPBELL.....	21st and Chestnut Sts., Op.
RADCLIFFE, McCLUNEY.....	1906 Chestnut St., Op.
REESE, WARREN S.....	230 S. 21st St., Op.
RIDPATH, R. F.....	1928 Chestnut St., ALR
ROMMELL, J. C.....	4501 N. Broad St., OALR
SARTAIN, PAUL J.....	2006 Walnut St., Op.
SCARLETT, HUNTER W.....	228 Locust St., Op.
SCHATZ, H. A.....	2035 Chestnut St., ALR
SCHNEIDEMAN, T. B.....	Professional Bldg., Op.
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